



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>



A

LANE

MEDICAL



LIBRARY

LEVI COOPER LANE FUND

—PRESENTED TO—

The New York Academy of Medicine.



By

The Society of the New York Hospital,

March, 1898.



Mr. S.

*J. J. Sabine
with the regards of
Francis Delafield*

STUDIES

IN

PATHOLOGICAL ANATOMY

BY

FRANCIS DELAFIELD, M.D.

ADJUNCT PROFESSOR OF PATHOLOGY AND PRACTICAL MEDICINE; VISITING PHYSICIAN AND CURATOR TO
BELLEVUE HOSPITAL; VISITING PHYSICIAN AND PATHOLOGIST TO THE ROOSEVELT HOSPITAL

VOLUME I.

PLATES I—XCIII.



NEW YORK

WILLIAM WOOD & COMPANY

1882

P

COPYRIGHT
WILLIAM WOOD & COMPANY
1882

WILLIAM WOOD & COMPANY

TROW'S
PRINTING AND BOOKBINDING COMPANY
201-213 East 12th Street
NEW YORK

5-1-1
D34
v.1
1882

PREFACE.

IN presenting to the profession the first volume of my "Studies in Pathological Anatomy," it seems proper to say a word concerning the scope and object of the work.

It has not been my intention to write a treatise on Pathological Anatomy, nor to give an account of the labors of others in the same field. My object has been a much more restricted one:—to describe and figure the minute lesions of disease from the material which has fallen under my own observation.

In doing this I have attempted to follow the purely objective method—to see and to describe whatever could be made out in the different post-mortem lesions of disease. Such a plan of study involves following Nature wherever she may lead, and gives rise to apparent contradictions, which cannot always be reconciled.

In such a descriptive work the drawings are of importance. It would, of course, be preferable to reproduce all the specimens by photography, but this plan seems to be only available for low magnifying powers. In this way I have employed the process for topographical purposes. The photographs have been made by Mr. Mason, and by the Artotype Company.

For high magnifying powers it is necessary to make drawings with the camera lucida, and these drawings should be of the actual

size of the specimens. They must also be reproduced without the intervention of an artist, even at the sacrifice of much beauty. The plates must, therefore, be of large size, and must be drawn directly on wood, stone, or copper, or reproduced by one of the photographic processes; all these plans have been tried with varying success. No one can feel more than I, how imperfect many of the plates are.

It is my intention to continue the work until I have described all the lesions with which I am acquainted; but the original plan of publishing in monthly parts will be abandoned. The fasciculi will be issued of varying size and at different intervals, according to the subjects treated of.

In the second volume the description of the lesions of chronic pulmonary phthisis will be completed.

FRANCIS DELAFIELD,

12 West Thirty-second Street.

STUDIES IN PATHOLOGICAL ANATOMY.

CONNECTIVE TISSUE.

THERE is a certain convenience in considering the human body as composed of connective tissue, mucous membranes, epithelial membranes, and viscera. Of these the viscera and the mucous membranes are not merely living tissues, but are capable of performing certain functions. The epithelial membranes and connective tissue simply act as a living framework, by which the viscera and mucous membranes are supported and held together.

To the connective-tissue group belong bone, cartilage, mucous tissue, neuroglia, and connective tissue proper; it is of the last of these that we have to speak.

Connective tissue proper covers the bones and holds their articular ends together, lines all the cavities of the body, forms a layer beneath the skin, separates the muscles from each other, joins them to the bones, covers the nerves, forms the walls of the blood-vessels, and makes part of the viscera. In these different situations it is called by different names, such as periosteum, ligaments, synovial membranes, serous membranes, subcutaneous connective tissue, fasciæ, tendons, neurilemma, interstitial tissue.

Wherever it is situated, connective tissue is composed of a basement substance and of cells, and imbedded in it are blood-vessels, lymphatic vessels, and nerves.

The basement substance, while still alive, is translucent and nearly homogeneous. After death it coagulates regularly into small fibres composed of still smaller fibrillæ. These fibres are arranged side by side so as to form lamellæ, or they cross and interlace with each other irregularly; they are then called fibrillated connective tissue. Or, the fibres may be so arranged as to form a network or reticulum; and they are then called reticulated connective tissue. In many places the basement substance is strengthened by small, smooth fibres, sometimes anastomosing with each other. These are called yellow elastic fibres, and seem to be of a different chemical composition from the rest of the basement substance.

The cells, excluding the wandering cells or white blood-globules, conform to two types: 1st, that of flat cells; 2d, that of cells with irregular branching bodies.

Wherever the basement substance is arranged in lamellæ and these lamellæ are joined together so as to form a membrane with a free surface, this free surface is covered by the flat cells. These flat cells have a large nucleus surrounded by a large, flat cell-body. The edges of the cells are in very close apposition, so that they adhere to each other. On the surfaces of the serous membranes, of the blood-vessels and of the lymphatic vessels these cells are called endothelial cells; they are also found, however, in the subcutaneous tissue and the fasciæ, although in these latter situations they have received but little attention. If we inject a solution of gelatine beneath the skin and allow it to harden, we find the subcutaneous connective tissue is separated into a great number of lamellæ enclosing irregular spaces. If we take the leg of a small animal, strip off the skin, soak it in a solution of nitrate of silver of the strength of 1 to 1,000, and then expose it to the light, we find the surfaces of these lamellæ covered by black lines enclosing regular spaces. These black lines mark the edges of the flat cells. If we prepare another leg in the same way, but soak it for a week in a solution of alcohol 100 parts, water 200 parts, 1 per cent. solution of osmic acid 1 part, then cut off small portions of the lamellæ and stain them with hæmatoxylin and eosin, we will see large, flat, nucleated cells covering the lamellæ; such cells are figured in Plate 1. In the upper part of the plate are figured

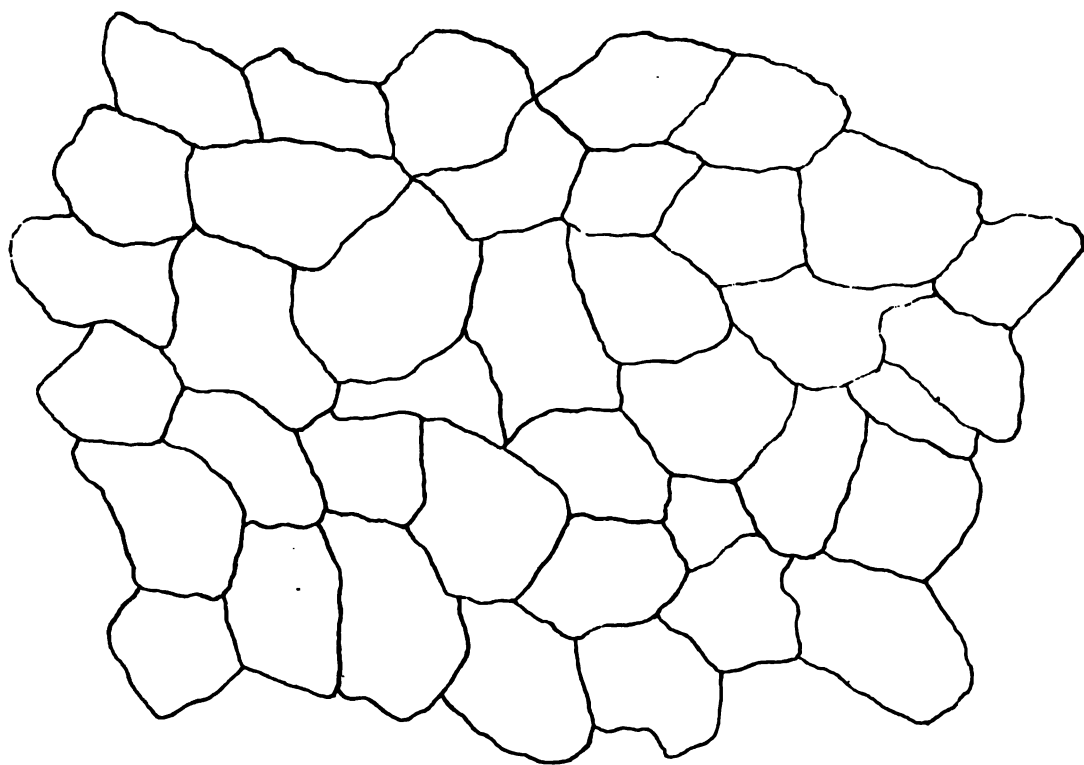


PLATE 1.

*Flat connective tissue Cells,
from the fascia of a dog's leg; magnified 750 diameters.*



the cells as shown by nitrate of silver. in the lower part the same cells as they appear when isolated.

The branched cells are found everywhere imbedded in the basement substance, or rather perhaps between the fibres of which the basement substance is composed. For the knowledge of the importance of these cells we are indebted to Virchow. He described them as fusiform or stellate cells with anastomosing processes, and gave them the name of connective-tissue corpuscles—a name which they have since retained. While he recognized their general shape and arrangement, he failed, owing to imperfect modes of preparation, to observe their full size and contours.

A few years ago Ranvier and Boll studied these cells more thoroughly, especially in tendons, and described them as flat cells. It seemed at first as if the older idea of connective-tissue corpuscles must be given up. Still further study, however, has shown that they are not flat cells, but are large irregular masses of protoplasm, with branching and anastomosing processes given off in all directions.

The nuclei of these cells are large, ovoid bodies, easily stained and easily seen, preserving their shape under most circumstances. One or more such nuclei are found in each cell. The cell-bodies, on the contrary, are composed of a very delicate substance, not easily stained and not easily seen. This substance coagulates into various shapes after death. As we look at the cell-bodies with the microscope, our idea of their shapes will vary with the completeness with which they are demonstrated. They will look like oval nuclei, or like narrow fusiform cells, or like flat, nucleated cells, or like large, irregular masses of protoplasm with processes running in every direction. The more complete the demonstration, so much the larger do the cell-bodies appear, and so much the more numerous are their anastomosing processes. They form a continuous system of cells imbedded in the basement substance. Their processes anastomose not merely in the same plane, but in every direction from the nucleus. Their branching shape resembles so closely that of the small lymph-spaces that the two have been supposed to be identical. But both cells and lymph-spaces can be demonstrated in the same specimen as distinct objects. Plate II. shows the branching

cells in the tendon of a dog's leg; Plate III. the cells in the omentum of the rabbit.

Connective tissue contains a very abundant system of lymphatics, consisting of canals of different sizes and of stellate spaces communicating with them. In it ramify also the blood-vessels and the nerves.

There are a number of changes which may occur in connective tissue called inflammatory changes, but they differ so widely in their anatomy that they must be described under separate names. We speak, therefore, of

1. Cellular Inflammation.
2. Inflammation with the production of Serum, Fibrine, and Pus.
3. Necrotic Inflammation.
4. Inflammation with the formation of Abscesses.
5. Reparative Inflammation.
6. Hyperplastic Inflammation.
7. Tubercular Inflammation.

By cellular inflammation I mean a change involving principally the fixed connective-tissue cells, these cells becoming increased in size and number. The blood-vessels and the basement substance undergo little or no change.

By inflammation with the production of serum, fibrine, and pus, I mean changes involving principally the blood-vessels. The plasma of the blood transudes and appears as fibrine and serum; the white globules emigrate and appear as pus-cells.

By necrotic inflammation, I mean changes involving both the blood-vessels and the tissue, and resulting in the death of the inflamed parts, either with or without putrefaction.

By inflammation with the formation of abscesses, I mean a complex process involving both the blood-vessels and the tissues, and resulting in the destruction of tissue, and the formation of pus.

By reparative inflammation, I mean the changes which result in the formation of new tissue, to take the place of tissues destroyed by injury or disease.

By hyperplastic inflammation, I mean an inflammatory growth of

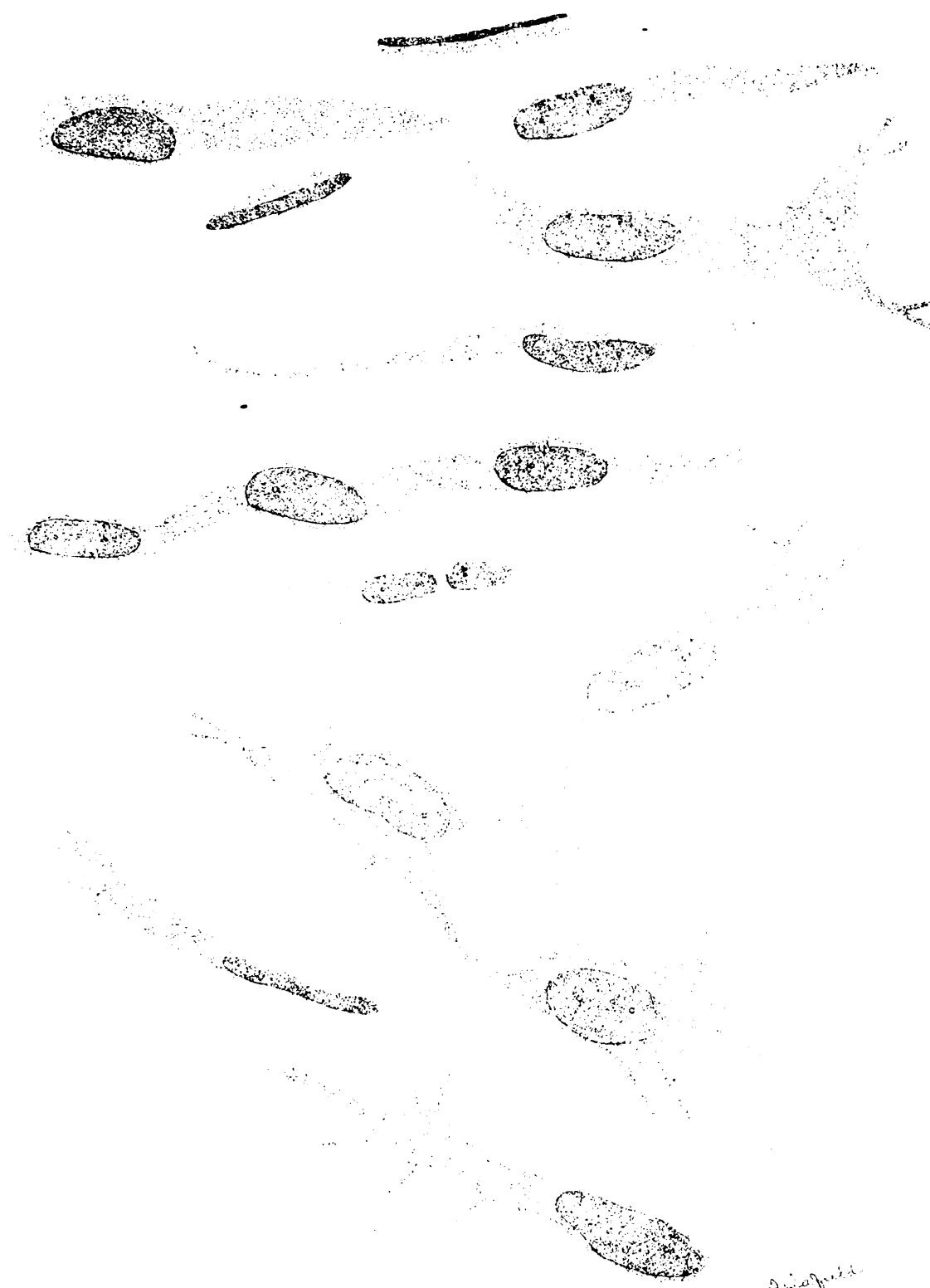


PLATE CC.

*Branching structure from Latta,
from the tendons of a developing, unmyelinated Nerve.*

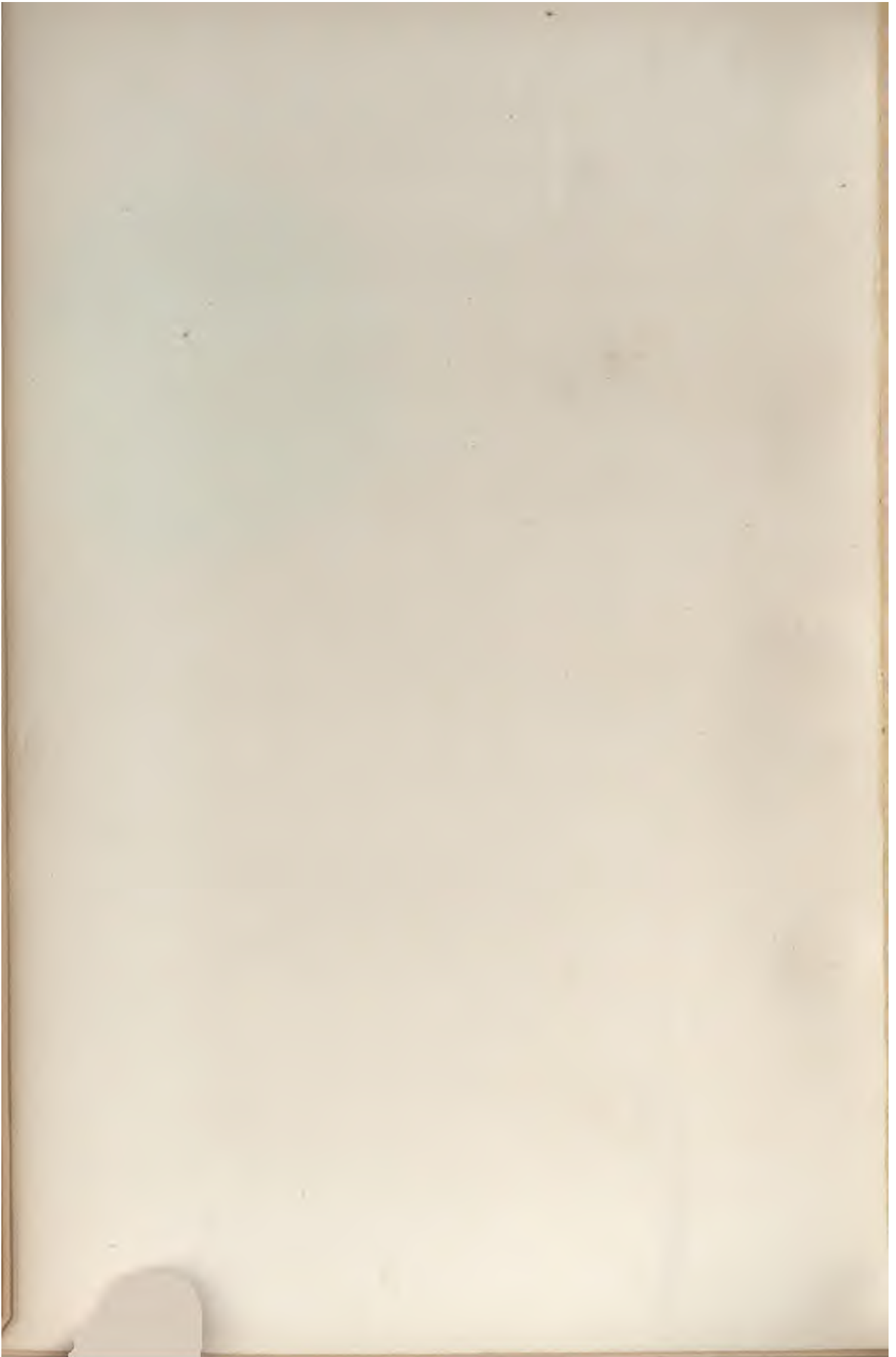
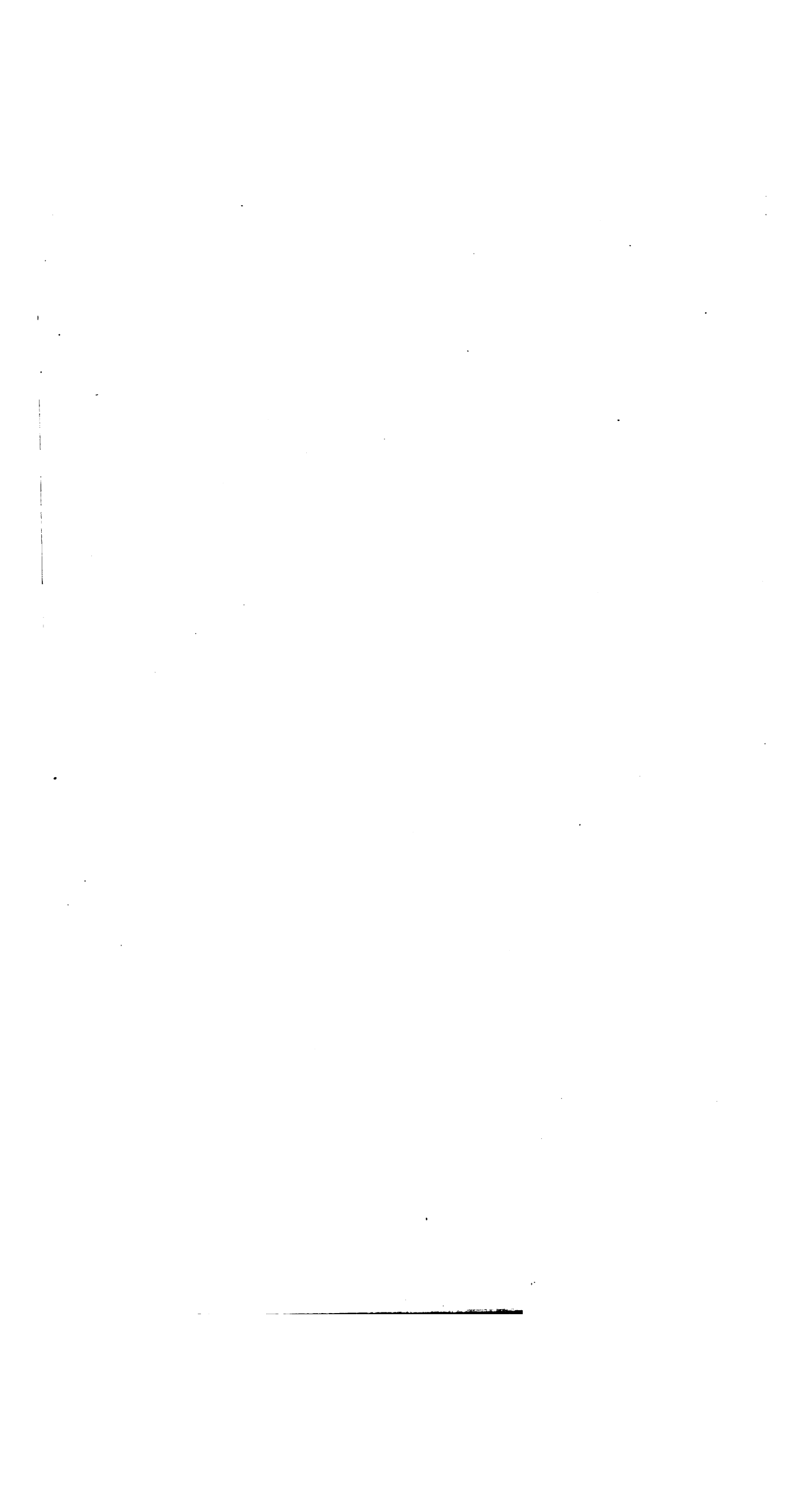




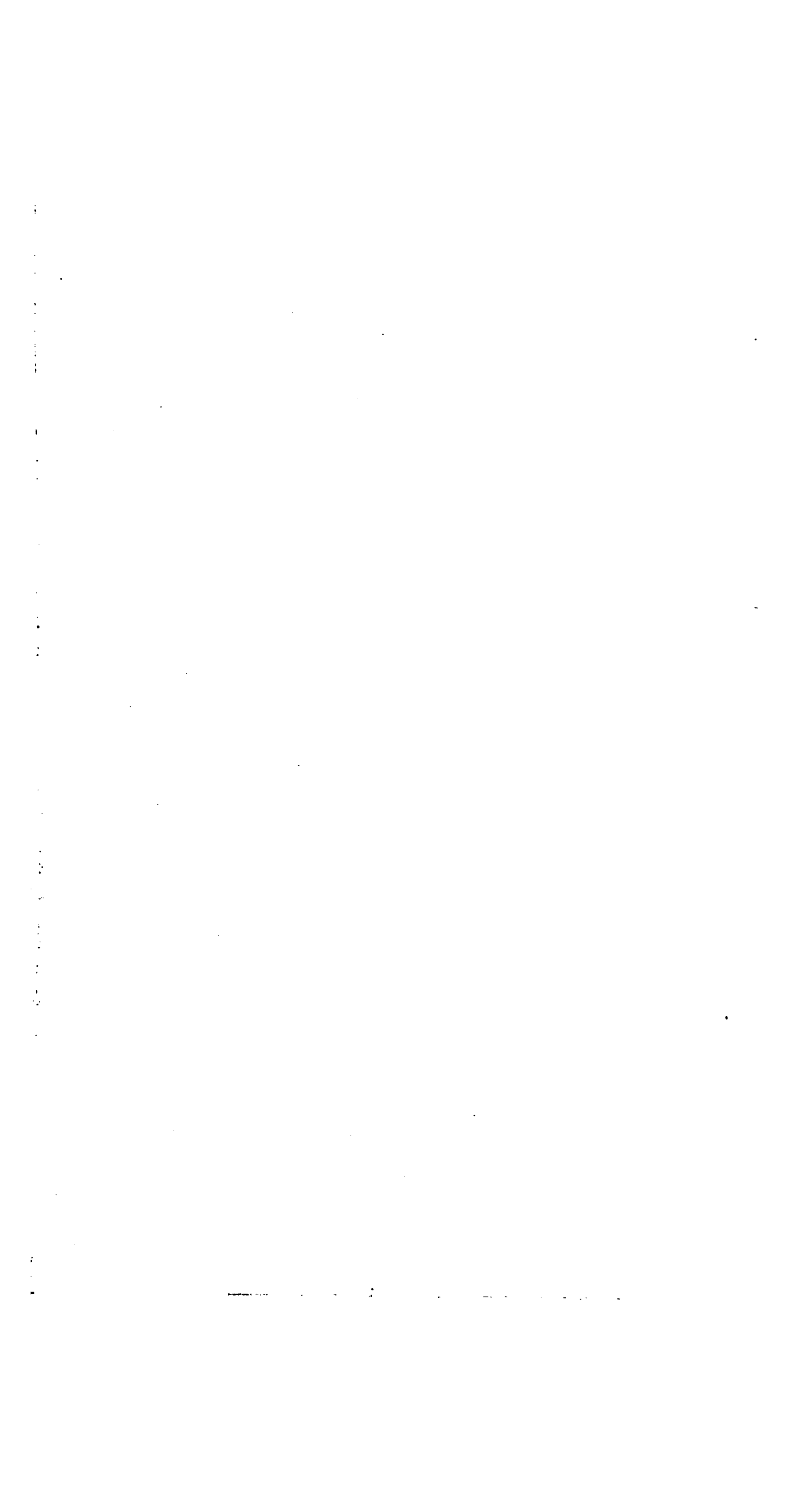
PLATE III.

*Branching connective tissue Cells
from the cementum of the rabbit magnified 150 diameters.*



new connective tissue, both cells and basement substance, not preceded necessarily by the production of pus or fibrine, or of cells alone.

By tubercular inflammation, I mean a form of inflammation resulting in the formation of an inflammatory product called tubercle. Tubercle is composed of a reticulated basement substance, of flat nucleated cells and of giant cells. It seems to be merely a form of connective tissue. It is produced either in the form of little rounded masses—tubercle-granula, or of a diffuse growth. Tubercle is a permanent inflammatory product. When once formed it remains unchanged or is transformed into ordinary connective tissue, or undergoes cheesy degeneration. Tubercular inflammation seldom, if ever, occurs by itself. It is accompanied by one or other of the varieties of inflammation, so that with the tubercle we find pus, fibrine, serum, new connective tissue, or necrotic changes.



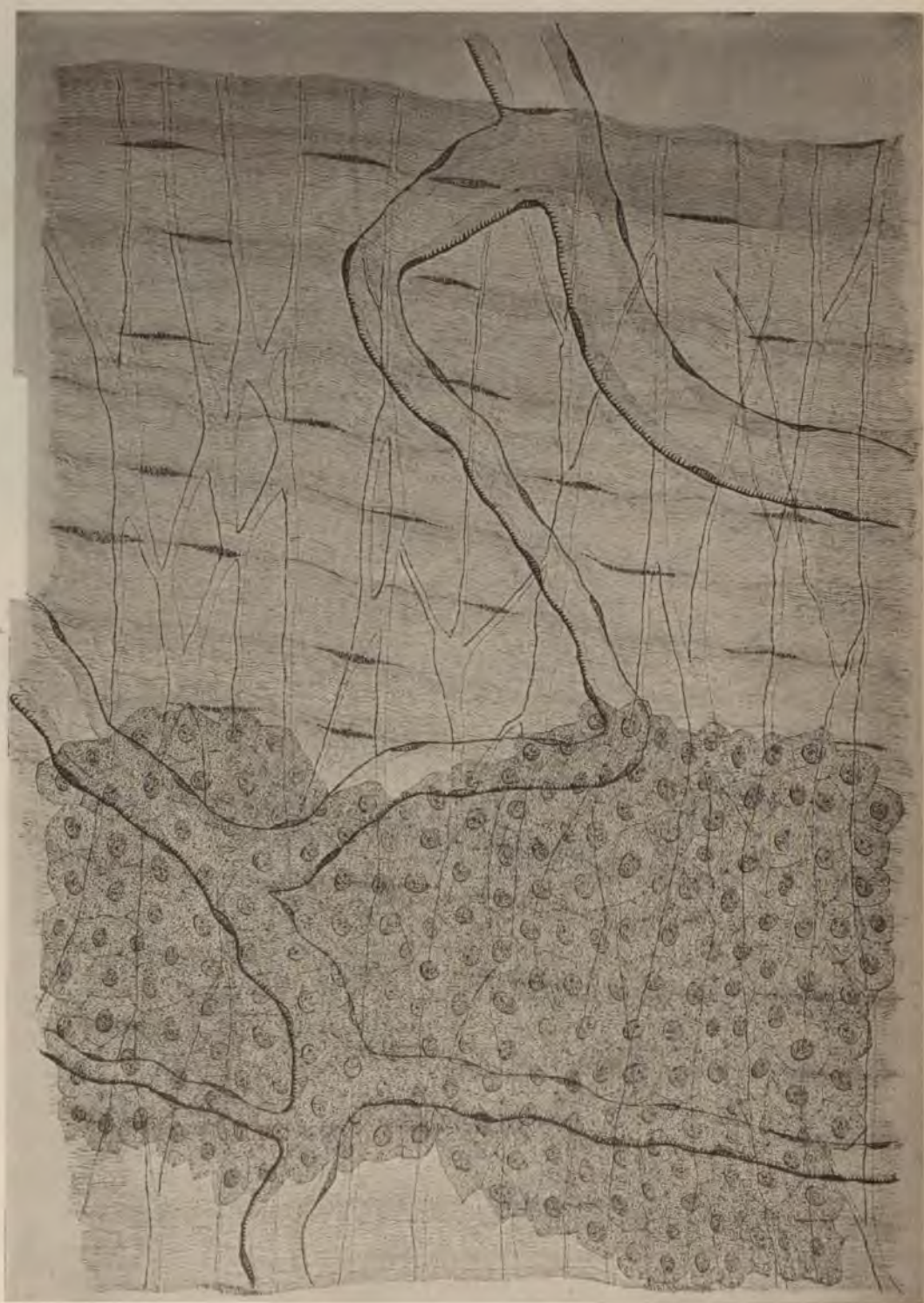


PLATE IV.

Pleura of the Dog; magnified 750 diameters.

THE PLEURA.

CONCERNING the anatomy of the pleura not much has been written. The most important contributions to our knowledge of it are by Dybkowsky and Klein.

Dybkowsky has described very fully the lymphatics of the pleura, especially those in the parietal portion of that membrane. His conclusions are in the main identical with my own observations.

Klein has described the endothelium and the lymphatics of the pulmonary and of the mediastinal pleura in a very satisfactory manner.

My own studies have been directed principally to that portion of the pleura which lines the chest-wall. Although in every pleurisy all the different parts of the pleura are usually inflamed, yet it is the parietal pleura which takes the principal share in the morbid process, and in idiopathic pleurisy it is the part first inflamed. The pleura is classed by anatomists with the serous membranes. The following general description of it is taken from Quain's "Anatomy:"

"The pleuræ are serous membranes, forming two shut sacs, quite distinct from each other, which line the right and left sides of the thoracic cavity, form by their approximation in the middle line the mediastinal partition, and are reflected each upon the root and over the entire free surface of the corresponding lung. Each pleura consists of a visceral and a parietal portion. The visceral portion (pleura pulmo-

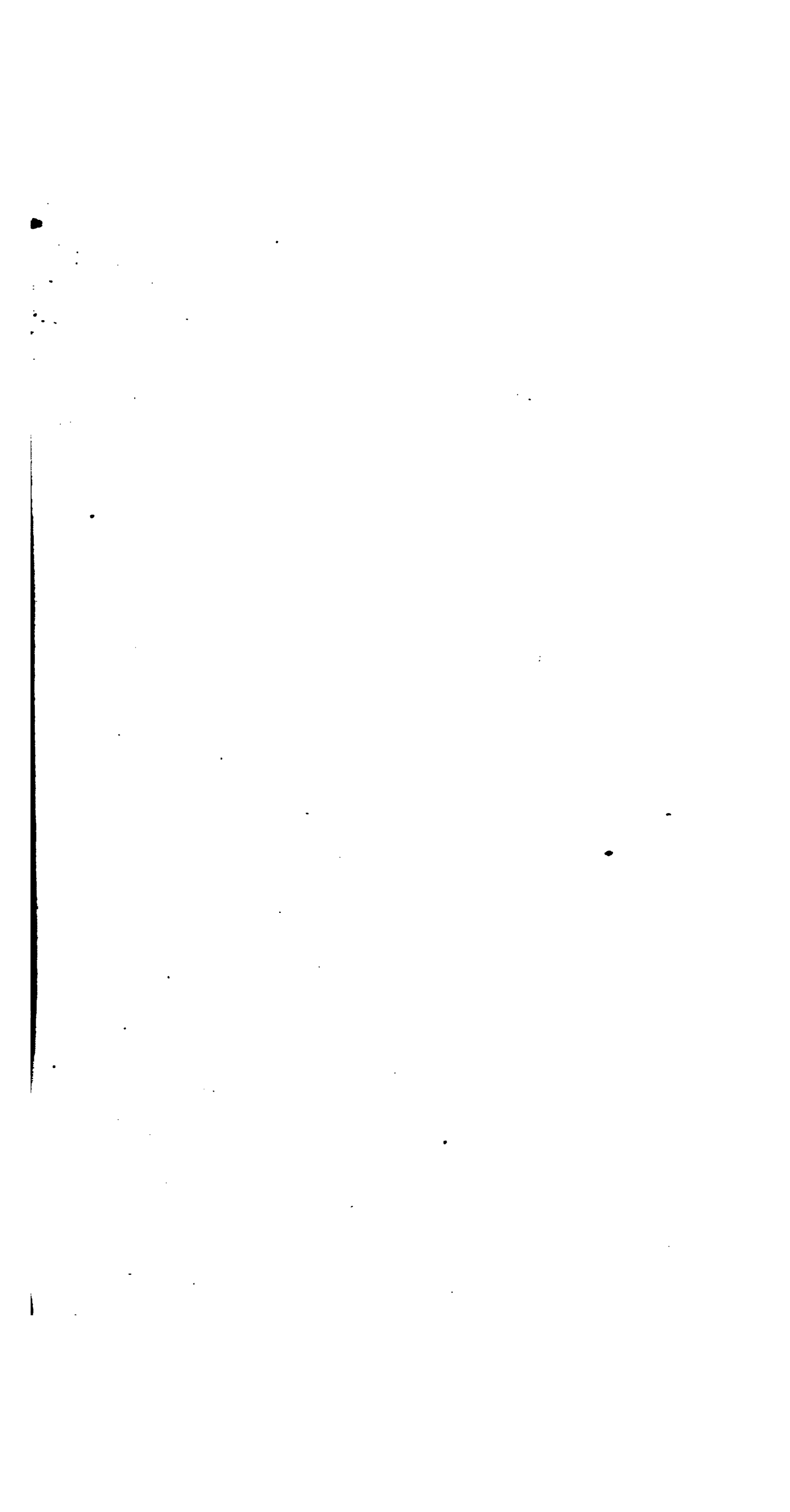
nalis) covers the lung; and the parietal portion lines the ribs and intercostal spaces (pleura costalis), covers the upper convex surface of the diaphragm, enters into the formation of the mediastinum, and adheres to the sides of the pericardium. At the root of each lung the visceral and parietal portions of the corresponding pleura are continuous with one another; and, at the lower border of the root, is a triangular fold of the serous membrane extending vertically along the inner surface of the lung down to the diaphragm, to which it is attached by its extremity; this fold is named *ligamentum latum pulmonis*.

"The upper part of the pleura rises into the root of the neck, reaching an inch or an inch and a half above the first rib, and passes up under cover of the *scaleni* muscles. The right pleura is generally stated to reach higher in the neck than the left, but this is certainly not always the case. Anteriorly the pleural sacs of opposite sides come nearly or altogether into contact behind the second piece of the sternum and continue so for some distance; but opposite the lower end of the sternum the right pleura passes beyond the middle line or remains close to it, while the left recedes to a variable distance. Inferiorly the pleuræ do not pass quite down to the attachments of the diaphragm, but leave a portion of its circumference in contact with the costal parietes. The right pleural sac is shorter and wider than the left."

Such is the usual description given of the pleuræ by general anatomists. For our purposes, however, it seems more convenient to consider it in a somewhat different manner.

The pulmonary pleura is not merely a membrane covering the lungs, but it is an essential part of those viscera. The lungs are composed principally of connective tissue, and this tissue is arranged on the surface of each lung in the form of a smooth membrane. It would be difficult for the lungs to be constructed at all without some such smooth external surface.

In the same way the inner surface of the thoracic cavity in which the lungs are contained is lined with connective tissue. There is not a membrane distinct from the tissues beneath, but a layer of connective tissue continuous with these tissues. The costal pleura is continuous with the periosteum of the ribs and the fasciæ of the intercostal





Reinhold

PLATE V.

*Endothelium and branching connective tissue cells,
from the pharynx of a dog, magnified 1300 diameters.*

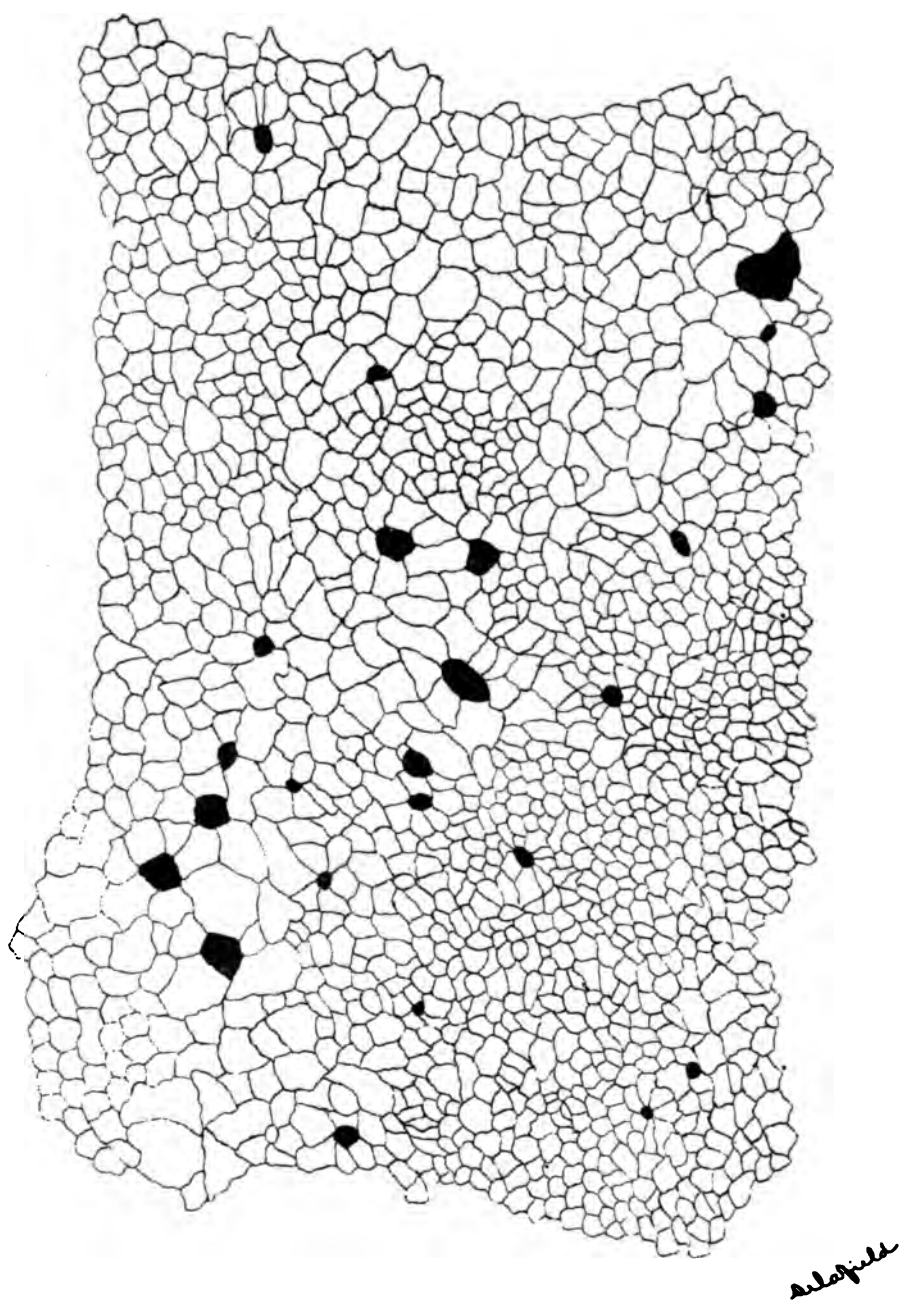


PLATE VI.

*Endothelium of Human Placenta;
magnified 90 diameters.*

muscles. The diaphragmatic pleura is continuous with the tendons and fasciæ beneath it. The mediastinal pleura forms a membrane by itself of somewhat different structure from the rest of the pleura.

The minute anatomy of the pleura may be conveniently studied in the dog and in man. In both the pleura is of sufficient thickness to be easily handled and split up into thin layers.

If now we cut out two or three ribs with the pleura in place, either from a dog or a human being, and allow them to soak for twenty-four hours in a mixture of dilute alcohol and osmic acid, we can with the forceps and scissors tear off one plane after another of connective tissue until we come down to the muscle and bone. The innermost of these planes, forming the free surface of the pleura, is covered with a continuous layer of flat nucleated cells, usually called endothelial cells. These cells, if demonstrated in this way, or with chloride of gold, or if looked at fresh in salt and water, look at first almost like a continuous nucleated membrane. The edges of the cells are in such close apposition that the lines of demarcation between the cell-bodies are not easily seen. Close inspection, however, with a high power shows that there really are dividing lines between the cells. If we soak the pleura for about twenty minutes in a solution of nitrate of silver (1-1000) and then expose it to the light, the silver will be oxidized in black lines between the edges of the cell-bodies. It is still uncertain whether these black lines indicate the existence of a cement substance uniting the cells, or whether the silver is precipitated between the edges of the cells.

When the endothelium is thus demonstrated with silver it becomes evident that all the cells are not of the same size, and that every here and there are small spaces where the oxidized silver forms black patches. These black patches are probably openings through the layer of endothelium into the lymphatics. These openings, or stomata, however, are not as regularly formed as they are in some other serous membranes.

In Plates IV. and V. is shown the endothelium of a dog's pleura; in Plate VI. the endothelium of the human pleura treated with nitrate of silver.

If now we carefully brush off the layer of endothelium, we find that

it was spread out over a smooth surface formed by the apposition of fibres of connective tissue reinforced by elastic anastomosing fibres. Running in parallel rows at the edges of the fibres are the fixed connective-tissue cells, which appear as bodies of ovoid or fusiform shape, as seen in Plate IV. More careful demonstration and a higher magnifying power show that the fixed cells really have a large cell-body with branching processes, as is seen in Plate V.

The deeper layers of the pleura are formed in the same way of planes of connective-tissue fibres, of anastomosing elastic fibres, and of branching cells, but the cells are most numerous in the layer just beneath the endothelium.

To demonstrate the blood-vessels of the pleura we can inject downwards through the carotid arteries, or upwards through the aorta. The capillaries show well after soaking in chloride of gold. The intercostal arteries break up into smaller branches terminating in a capillary plexus with wide meshes. Some of the capillaries extend into the layer of connective tissue immediately beneath the endothelium. The vessels are surrounded by a layer of branching connective-tissue cells.

The lymphatics can be shown either by soaking the pleura in a solution of nitrate of silver (1-1000), or by injecting a five per cent. solution of Prussian blue into one of the pleural cavities about two hours before the animal is killed.

There are a great number of anastomosing lymphatic vessels of large size lined with endothelium, and apparently communicating with these vessels are irregular stellate spaces. The lymphatic vessels are only found over the intercostal spaces; they do not extend into the pleura which covers the ribs. In Plate VII. are seen the lymphatics of a dog's pleura, and it will be seen that the lymphatics occupy a larger area than does the tissue between them.

The lymphatic spaces do not correspond to the branching connective-tissue cells, but are distinct from them. Both can be demonstrated in the same specimens, as is seen in Plate VIII.



PLATE VII.

*Lymphatics of the Parietal Pleura of the Dog;
magnified 90 diameters.*



PLATE VIII.

*Lymphatic Spaces and connective tissue Cells,
from the Pleura of a Dog; magnified 750 diameters.*

THE INFLAMMATIONS OF THE PLEURA.

THE human pleura may become the seat of a variety of inflammatory processes, which we will describe under the following names :

(1.) Pleurisy with the production of fibrine alone. Dry pleurisy. *Pleuritis sicca*. Acute pleurisy.

(2.) Pleurisy with the production of fibrine and serum. Pleurisy with effusion. Subacute pleurisy.

(3.) Pleurisy with the production of pus. Empyema.

(4.) Dropsical effusions in the pleural cavities. Hydrothorax.

(5.) Chronic pleurisy with permanent adhesions.

(6.) Pleurisy of chronic pulmonary phthisis.

(7.) Tubercular pleurisy.

1. Pleurisy with the production of fibrine.

This variety of pleurisy occurs after exposure to cold, after fracture of the ribs, with pneumonia, with phthisis, with infarctions of the lungs, in the course of pyæmia, of puerperal fever, of peritonitis and of Bright's disease. Some individuals have a peculiar predisposition to the disease, and will suffer from repeated attacks during successive winters.

In the idiopathic cases the inflammation usually involves circumscribed portions of the costal, pulmonary, mediastinal, or diaphragmatic pleura. Sometimes, however, a larger area of pleura is involved; and, as an exceptional thing, the entire pleura of one side is inflamed.

Such cases usually die (if they die at all) after the inflammation is fairly established, or else after it has run its course. When the inflammation is fairly established, we find the affected portions of the pleura covered with a thin layer of fibrine, while the pleura beneath is congested. I have seen one case in which the amount of fibrine was so great that the lung was compressed by it against the vertebral column. After the inflammation has run its course, we find new connective tissue thickening the pleura and forming adhesions between the lobes of the lungs, and between the lungs and the chest wall.

2. Pleurisy with the production of fibrine and serum.

This form of pleurisy is due to the same causes as the form just described. It usually involves the entire pleura of one side, sometimes of both sides. If the pleurisy is double, it is often complicated by pericarditis.

While the inflammation is in progress we find most of the pleura covered with a thick layer of fibrine. This fibrine varies in color and consistence according to the number of pus-globules entangled in it. It not only coats the pleura, but it forms adhesions between the pulmonary and costal pleura. In the pleural cavity is serum in variable quantity. This serum is almost clear, or turbid from the admixture of pus-cells and flocculi of fibrine. The lung is compressed against the vertebral column by the fluid, or the adhesions fasten it to the chest wall in different positions.

The patients may die during this stage of the disease; or the inflammation may change its character and become purulent; or the patients recover with permanent adhesions and thickenings of the pleura.

These two forms of pleurisy, although differing widely in their clinical history, are yet essentially the same. In both of them we find a regular sequence of changes. First, the production of fibrine and a few pus-cells either with or without serum. Second, a gradual absorption of the serum, and a disappearance of the fibrine. Lastly, as the regular termination of the inflammation, the formation of permanent new connective tissue in the shape of adhesions or of thickenings of the pleura. Variations from the regular course of the inflammation are effected by the excessive formation either of the fibrine, the pus, or the



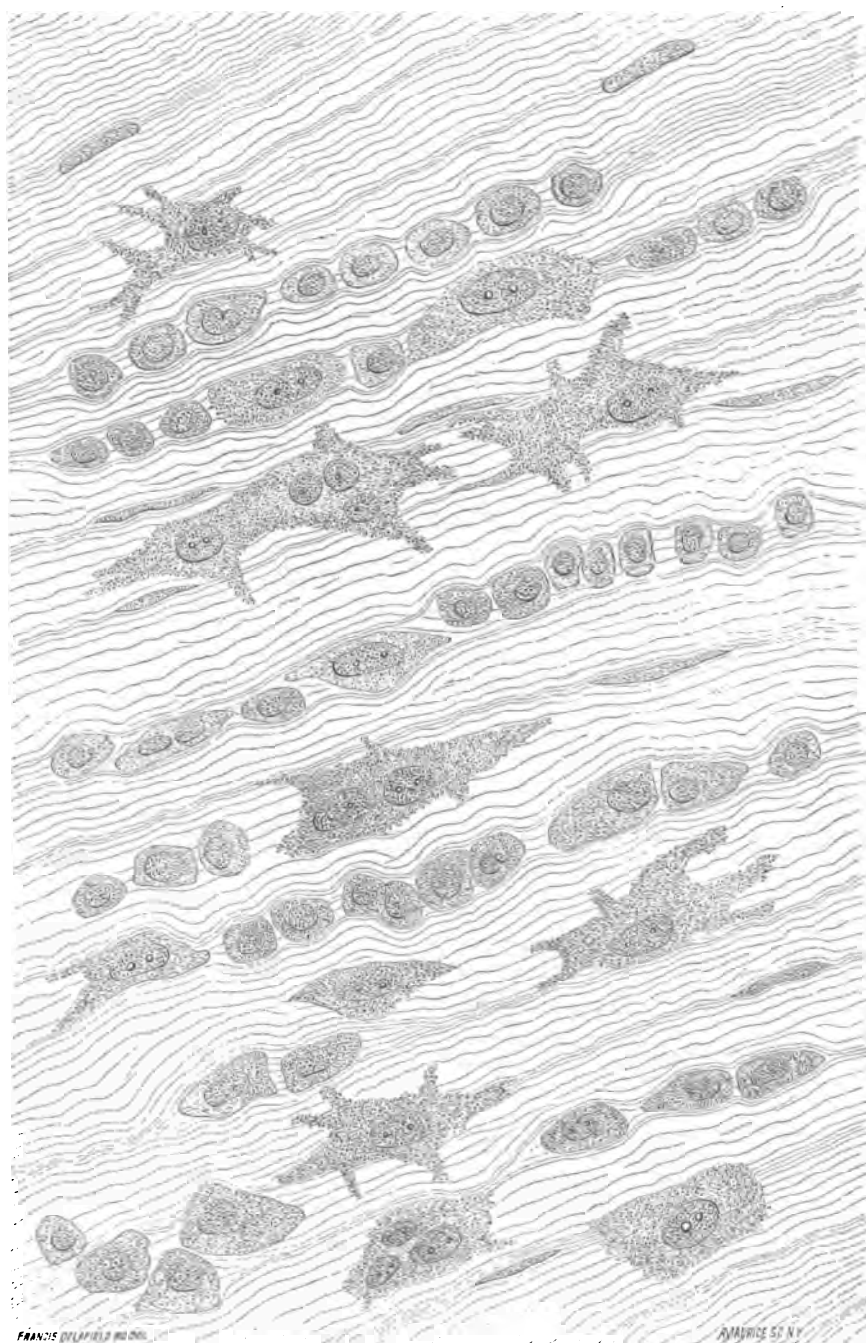


PLATE IX.

*Pleurisy of the Dog, 24 hours. New Cells;
magnified 750 diameters.*

serum, and by the failure of the absorption of these inflammatory products.

If we endeavor to follow out the successive changes by which the fibrine, pus, and serum make their appearance and then disappear, and the way in which permanent new tissue takes their place, we encounter several difficulties. It is impossible, in any ordinary experience, to obtain autopsies which will give the lesions belonging to each successive day of the disease; the pleura does not really show well if the patient has been dead more than two or three hours before the autopsy; and in most cases the inflammation is too energetic, its products are too abundant to be easily studied.

To obviate these difficulties we must resort to the lower animals. By injecting a saturated solution of chloride of zinc, with a hypodermic syringe, into the pleural cavity of a dog, we can excite a pleurisy exactly resembling what we see in the human subject. By varying the amount of fluid injected, and by injecting it into one or both pleural cavities, we can obtain pleurisies of different degrees of intensity, and with different amounts of products of inflammation. By using a number of animals we can observe the course of the inflammation from hour to hour, and from day to day.

The very first change we observe in such an artificial pleurisy is a simple congestion. The parietal pleura is of a uniform rosy red color, its surface is shining and moist as in health. There is no serum and no fibrine. Minute examination, however, shows that the endothelial cells have fallen off in patches, and that where they have thus fallen off the superficial connective-tissue cells are swollen and multiplied, and there are some pus-cells. This is all that takes place for from half an hour to six hours after the injection has been made.

The next step in the inflammatory process is the appearance of serum and fibrine in variable amounts. The serum collects in the bottom of the pleural cavity. It is usually slightly turbid from the admixture of blood-globules and pus-cells. The fibrine coats the whole surface of the pleura. There may be so little of it that it cannot be seen with the naked eye, or its amount may be considerable. The surface of the pleura loses its natural glistening appearance in proportion

to the amount of fibrine coating it. The fibrine appears first in the form of little knobs and strings, or of a thin layer of granular matter. Where the endothelium still adheres the edges of the cells are separated from each other by granular matter. A few pus-cells are seen entangled in the fibrine, and imbedded in the pleura. The swelling and new growth of the fixed connective-tissue cells is now very marked. The branching bodies of the old cells are readily seen. The new cells are of different shape from the old ones. They are polygonal cells with a distinct cell body and nucleus, and are arranged in rows parallel with the course of the fibres. These new cells give the impression of being formed in some way from the old fixed cells, but precisely how I am unable to say. The time varies from half an hour to seven hours before these changes take place. By the end of twenty-four hours they are always fully developed.

Plate IX. shows the swollen connective-tissue cells, and the new cells in the superficial layer of the pleura, just beneath the fibrine, the latter being stripped off.

On the second day of the pleurisy the gross and minute appearances are unchanged, only the layer of fibrine is thicker and there is more serum. Up to this time the lymphatic vessels and spaces can still be demonstrated with nitrate of silver, and are apparently empty.

On the third day in some cases, not until the fourth in others, the growth of new cells has become well marked. The layer of fibrine on the pleura is more consistent, the serum is still abundant or has begun to disappear. The new cells present a great variety of shapes. Some are of the shapes seen in Plate IX., others are of the same shape but smaller, others are much larger and of polygonal, rounded, fusiform, or branching shape. These cells are very abundant in the superficial layers of the pleura, but not in the deeper layers. The pleura is not much thickened. They are also present in large numbers entangled in the layer of fibrine coating the pleura, and in the adhesions of fibrine between the pulmonary and costal pleura.

Plate X. shows such a layer of fibrine stripped off from the pleura with large numbers of new cells imbedded in it.

By the fourth or fifth day, according to the case, new blood-vessels

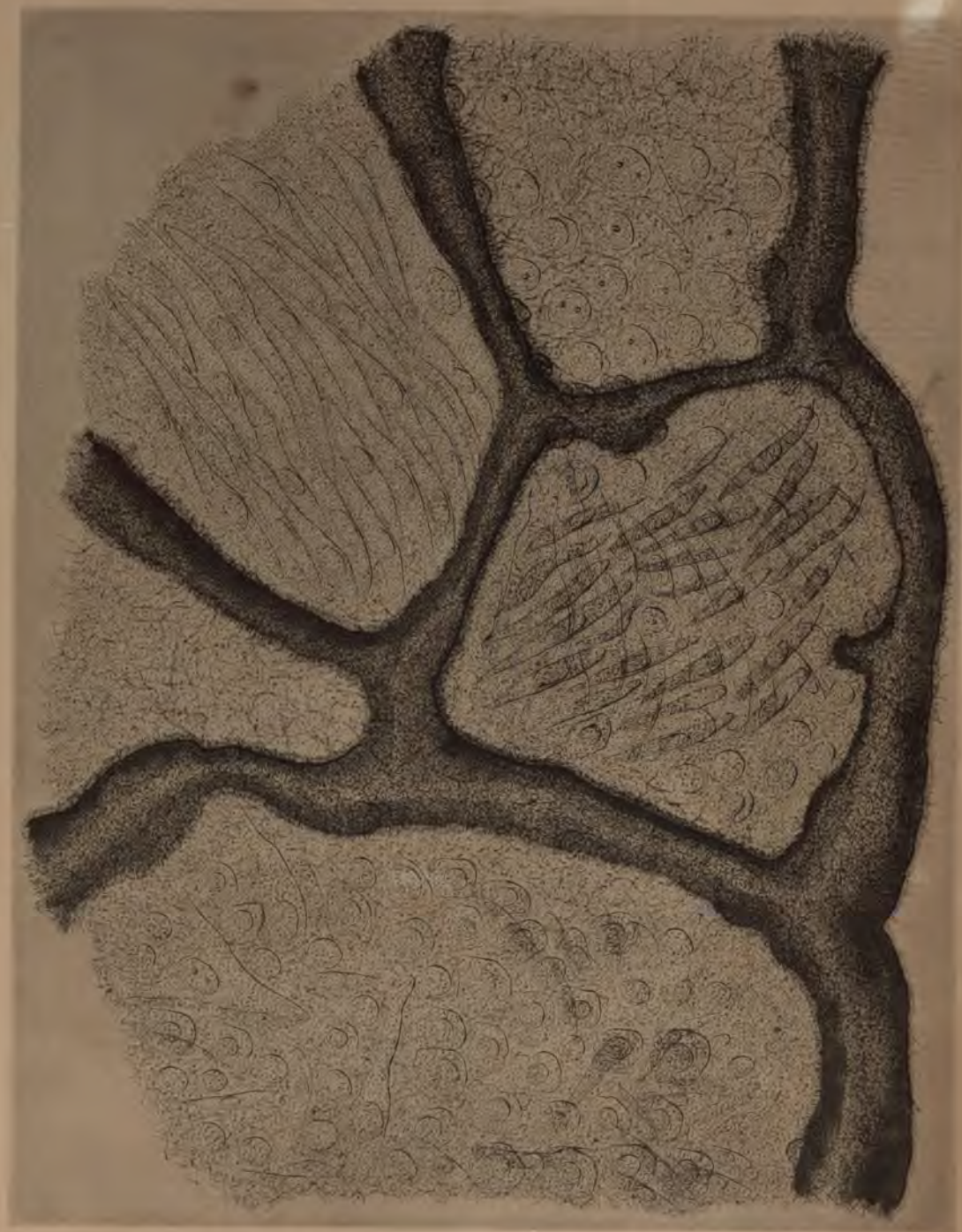


PLATE X.

Pleura of the Dog. 3d day.
Layer of Cells and Fibrous. 750 diameters.



PLATE XI.

*Pleura of the Dog, 5th day. Layers of Viscous Tissues;
magnified 750 diameters.*

make their appearance in the layer of fibrine and cells. These vessels can be injected artificially from the aorta; they are continuous with the old vessels of the pleura. They are of large size, numerous, and with thin walls. In the pleura itself the vessels are increased in size and number. At the same time the fibrine begins to disappear, and the new cells form a more continuous layer.

Plate XI. represents a portion of the layer of new tissue stripped off from the pleura. The vessels are distorted by the pressure of the artificial injection. The cells form a continuous layer, the fibrine at this place having disappeared.

By the sixth day the layer of fibrine and new tissue on the surface of the pleura is more adherent. It begins to form a part of the pleura. The serum has by this time usually disappeared; the fibrine also is less in amount. The new cells are still numerous, and they are becoming arranged into two divisions: a superficial layer resembling the old endothelium, and a deeper layer of fusiform and branching cells. Of the new endothelial cells some present a curious appearance from the distention of the cell-bodies by cavities or vacuoles.

On the seventh day the condition is much the same. The new endothelial cells appear first in patches and rows, which afterwards increase in area. At this time the adhesions between the costal and pulmonary pleura are also partly covered with endothelium.

After this time the fibrine continues to diminish in amount until by the fourteenth day it has usually entirely disappeared. Of the new cells, the superficial layer gradually forms a continuous layer of endothelium. In the deeper layer the rounded and polygonal cells are not as numerous, while there are many large and small fusiform and branching cells, some of them very long and continuous with each other, as seen in Plate XII. Gradually a basement substance makes its appearance between the cells, at first homogeneous, afterwards fibrillated.

These changes continue until by the end of three weeks the pleura has its normal appearance, except that it is thickened, and that there are adhesions between the costal and pulmonary pleura. But if we examine the superficial layers of this thickened pleura we will still find an excess of cells.

The lesions of human pleurisy are substantially the same as those just described. The only difference is that the fibrine, pus, and serum are produced in very much larger quantities, and as a result of this the absorption of these products and the formation of new tissue follow more slowly. It may also happen that the inflammation changes its character and becomes purulent.

We may then draw the following conclusions :

1. In pleurisy with the production of fibrine alone, or of fibrine and serum, two distinct pathological processes regularly take place.
(a) The blood-vessels become congested and through their walls transude the plasma of the blood and a few white blood-globules; part of the plasma appears as serum, part of it contains substances which by the action of the emigrated white globules are transformed into fibrine.
(b) At the very commencement of the inflammation the superficial fixed connective-tissue cells also begin to increase in size and number.

2. The products of the first of these processes—the fibrine and serum—are regularly reabsorbed. In many patients, however, the course of the disease is irregular, and these products remain for a long time.

3. The product of the second of these processes regularly increases. The new cells formed in the superficial layers of the pleura wander into the fibrine coating the pleura and forming adhesions, and there grow and multiply. New blood-vessels and a new basement substance are formed between these cell

4. The natural termination of such a pleurisy is the formation of new tissue coating the pleura and forming adhesions.

5. The irregular terminations of such a pleurisy are: the death of the patient; the protracted existence of the fibrine and serum; the change into empyema; and the formation of an excessive amount of new connective tissue.

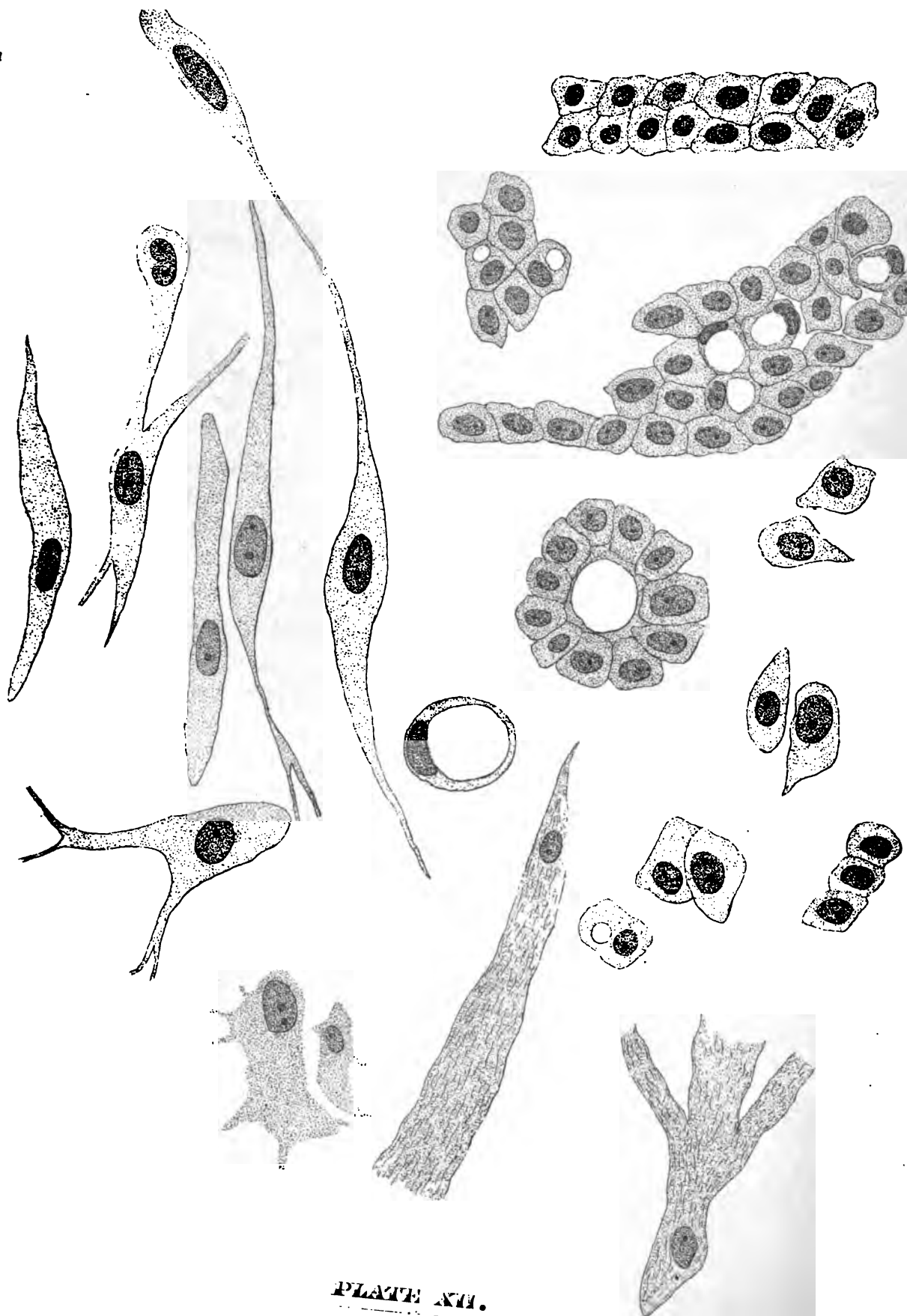


PLATE XII.

*Placenta of the dog 9 days.
New endothelial and connective tissue cells,
magnified 750 diameters.*

EMPHYEMA:

Pleurisy, with the production of fibrine, serum, and a large amount of pus, occurs under several different conditions.

1. A person may be attacked by a pleurisy in which there is from the first an undue amount of pus with the fibrine and serum. In such a case the constitutional disturbance is very severe. These cases are fortunately not common.

2. The pleurisy at first is only attended with the production of fibrine and serum, and after the lapse of weeks or months gradually assumes a purulent character; or, instead of the change in the character of the inflammation being gradual, it is sudden, with a corresponding change in the constitutional symptoms.

3. Abscesses in the wall of the thorax, or in the liver, or in the abdomen, rupture into a pleural cavity and set up a purulent inflammation.

4. Abscesses of the lung, or phthisical cavities in the lung, rupture into the pleural cavities and produce empyema.

5. In the course of pyæmia the pleura may become the seat of purulent inflammation.

6. The inflammation may be not only purulent, but gangrenous. The fluid in the pleural cavity, the fibrine and pus coating the pleura, and the pleura itself, may putrefy with the production of bacteria and the evolution of gases. This may take place either in a closed pleura, or in one which has been opened by incision.

7. If there is a permanent opening either in the lung or in the chest-wall, there is air in the pleura in addition to the inflammatory products. Such a condition is called pyopneumothorax.

In all these different cases the pleural cavity is partly or completely filled with purulent fluid, and the lung is either compressed against the vertebral column, or partly adherent to the chest-wall. Occasionally, however, the purulent fluid is shut in by adhesions either between part of the lung and the thoracic wall, or between the lung and the diaphragm, or between the lung and the pericardium, or between the lobes of the lung.

The gross appearance of the purulent fluid is not like that of the pus of an abscess, but is thinner and has more the character of a purulent serum. It seems to be composed of serum, such as is produced in a pleurisy with effusion, of pus-globules, and of larger cells looking like altered endothelium. According to the number of pus-globules in proportion to the serum, the fluid is thicker or thinner. The pus-globules are some of them well formed, with a spherical body containing two or three nuclei; others have a single large nucleus surrounded by a narrow polygonal cell-body; others are fatty or partly broken down.

In these different clinical varieties of empyema it will be observed that the inflammation may be from the outset a purulent one, as in the sets of cases marked 1, 3, 4, 5; or it may be first a pleurisy with the production of fibrine and serum, and the pus appears later.

I am unable to say what are the first changes in a pleurisy which has a purulent character from the outset. Patients do not die until the disease has lasted for a number of days. It is difficult to produce empyema in dogs, and when produced one cannot be sure that the early stages are identical with what occurs in the human subject.

The best way I have been able to find of producing empyema in the dog is to tie a string around one of the ribs. This acts as a seton and excites suppurative inflammation along its track; sometimes also the sinus thus formed allows air to enter the pleural cavity.

In such an empyema the first change to be noticed is that the bodies of the endothelial cells become shrunken in such a way that they look as if processes were given off from them. At the same time pus-globules make their appearance in the pleura just beneath the endothelium. Such a condition is seen in Plate XIII. During the first twenty-four hours no other changes were observed. There was no fibrine, no serum, no increase of fixed connective-tissue cells.

By the second day there is a little purulent serum in the pleural cavity, but no fibrine on the pleura. The endothelium is now still more altered. Large vacuoles are formed in the bodies of many of the cells, and the other cells are compressed and misshapen. There are more pus-cells in the pleura, and the fixed connective-tissue cells are moderately increased in size and number.

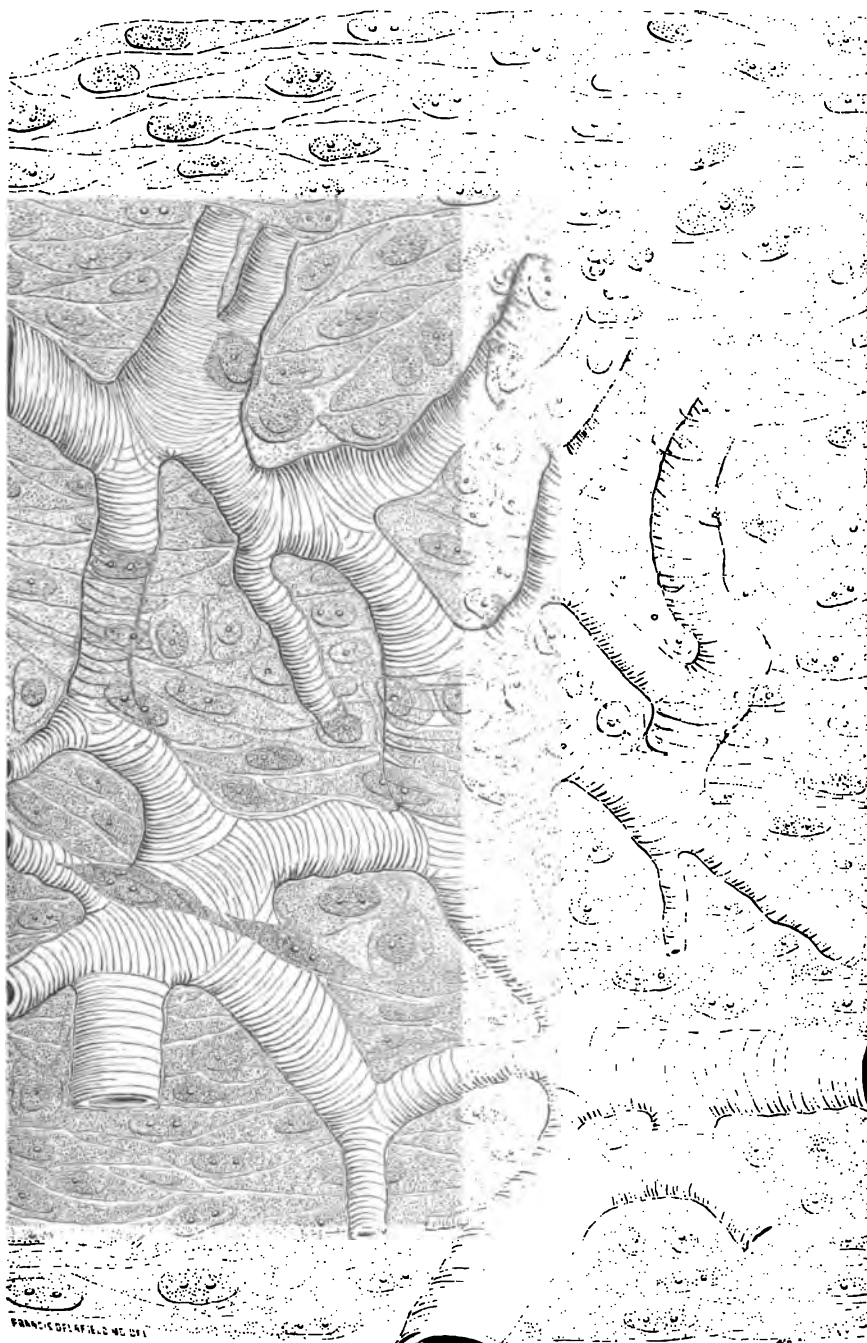


PLATE XI.

*Placenta of the Dog, 5th day. Layers of New Tissues;
magnified 750 diameters.*

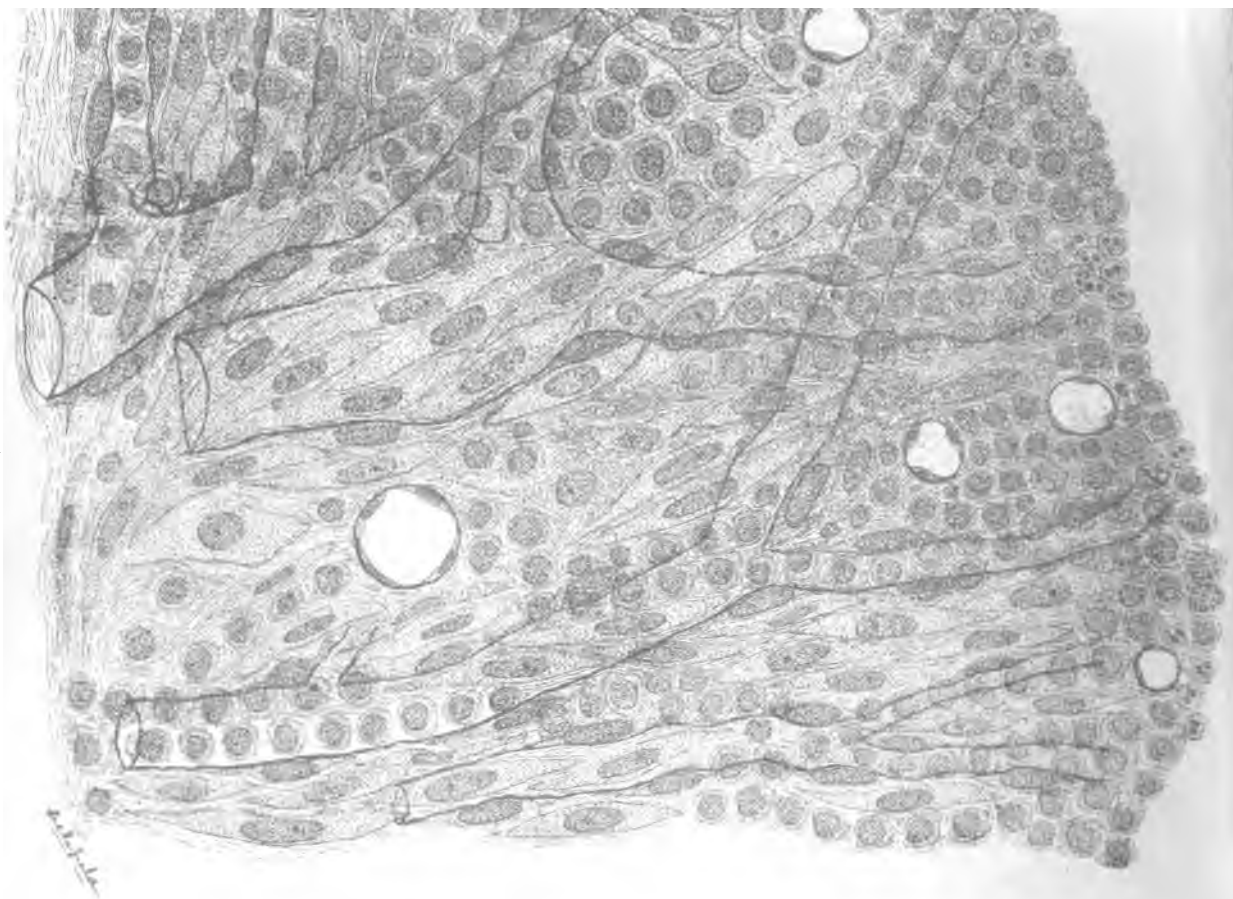


PLATE XIV.

*Empyema of ten days duration in a dog.
Vertical section, unguished 1/30 diameter.*

After the second day the amount of purulent serum increases. The vacuolated condition of the endothelium is more marked, and the vacuoles contain pus-globules. The pleura becomes thicker and thicker; more pus-globules accumulate in it; and the connective-tissue cells continue to increase in number. The blood-vessels of the pleura are dilated and appear more numerous.

By the tenth day the surface of the thickened pleura has become uneven, looks like granulation tissue, and is covered with pus. The superficial layers of the thickened pleura are formed of small polygonal cells, with large nuclei, arranged in a reticulated basement substance. There are numerous large blood-vessels with thick walls. Plate XIV. represents a vertical section of the inner layer of the pleura in the empyema of a dog on the tenth day.

It is not until this stage of the inflammation is reached that the gross appearance of the artificial empyema looks like that of the empyema of the human subject, so that it is doubtful how far the early changes just described correspond with the early stages of human empyema.

If, however, the pleurisy is at first a pleurisy with effusion, and afterwards becomes purulent, we have more opportunities of seeing the different stages of the inflammation. In the earlier stages the serum is still thin, but yellow from the admixture of pus-globules; the surface of the pleura is thickly coated with fibrine, and in the fibrine are some pus-cells and but few new connective-tissue cells. There is a growth of new connective-tissue cells on the surface of and in the superficial layer of the pleura. After the empyema has lasted somewhat longer, the layer of fibrine remains the same, but the pleura itself is thicker, and this thickening is not due to a layer of new tissue on its surface, but to a cell-growth through its whole thickness. The new cells resemble the new polygonal cells seen in Plate IX., but they are smaller and more numerous. They split up the basement substance and present such an appearance as is seen in Plate XV., which represents a vertical section of the human pleura from a case of empyema of some weeks' duration.

When the empyema has lasted for several weeks or months, the condition of the pleura is the same whether the inflammation was from the

outset purulent, or whether it succeeded a pleurisy with effusion. The pleural cavity contains purulent serum. The pleura itself is thickened, sometimes as much as half an inch. The surface of the pleura is in some cases coated with fibrine, in other cases it is bare of fibrine and looks something like the surface of an old ulcer of the leg. If we make vertical sections of the pleura we find in its deeper layers such an appearance as is seen in Plate XV.; the fibres split up by small polygonal cells, the number of the cells varying in different cases. In the superficial layers of the pleura the cells are very much more numerous. They are of the same polygonal shape, and are so close together as to obscure the basement substance. There are a few pus-cells mixed with them. The fibres of the basement substance are not parallel with the fibres of the deep layers of the pleura, but run vertically to these and may form a fine reticulum. In the most superficial cells the cell-bodies may be wanting, and the nuclei may be fatty. There is a very abundant supply of large blood-vessels with thin walls. Such a vertical section is seen in Plate XIV., which is taken from the artificial empyema of a dog on the tenth day. The plate represents equally well the appearance of any old human empyema.

In this condition the pleura may remain for months or years, its inner layers continuing to be formed of this same arrangement of small cells, scanty basement substance, and large blood-vessels, while the deeper layers are formed of a dense fibrous tissue with comparatively few cells.

Many of the patients die with the pleura in this condition; some from exhaustion, some from inflammations of the lungs, some from gangrene of the pleura, some suddenly from unknown causes.

In some old cases the inflammation will extend to the perichondrium of the cartilages and the periosteum of the ribs and cause necrosis of these; or there may be ossifying periostitis with thickening of the ribs.

In some cases the inflamed pleura reaches an enormous thickness, and is infiltrated with the salts of lime, so that calcareous plates are formed in it.

In the cases which recover the formation of pus ceases. The surface of the pleura becomes clean and of a reddish color, looking like a

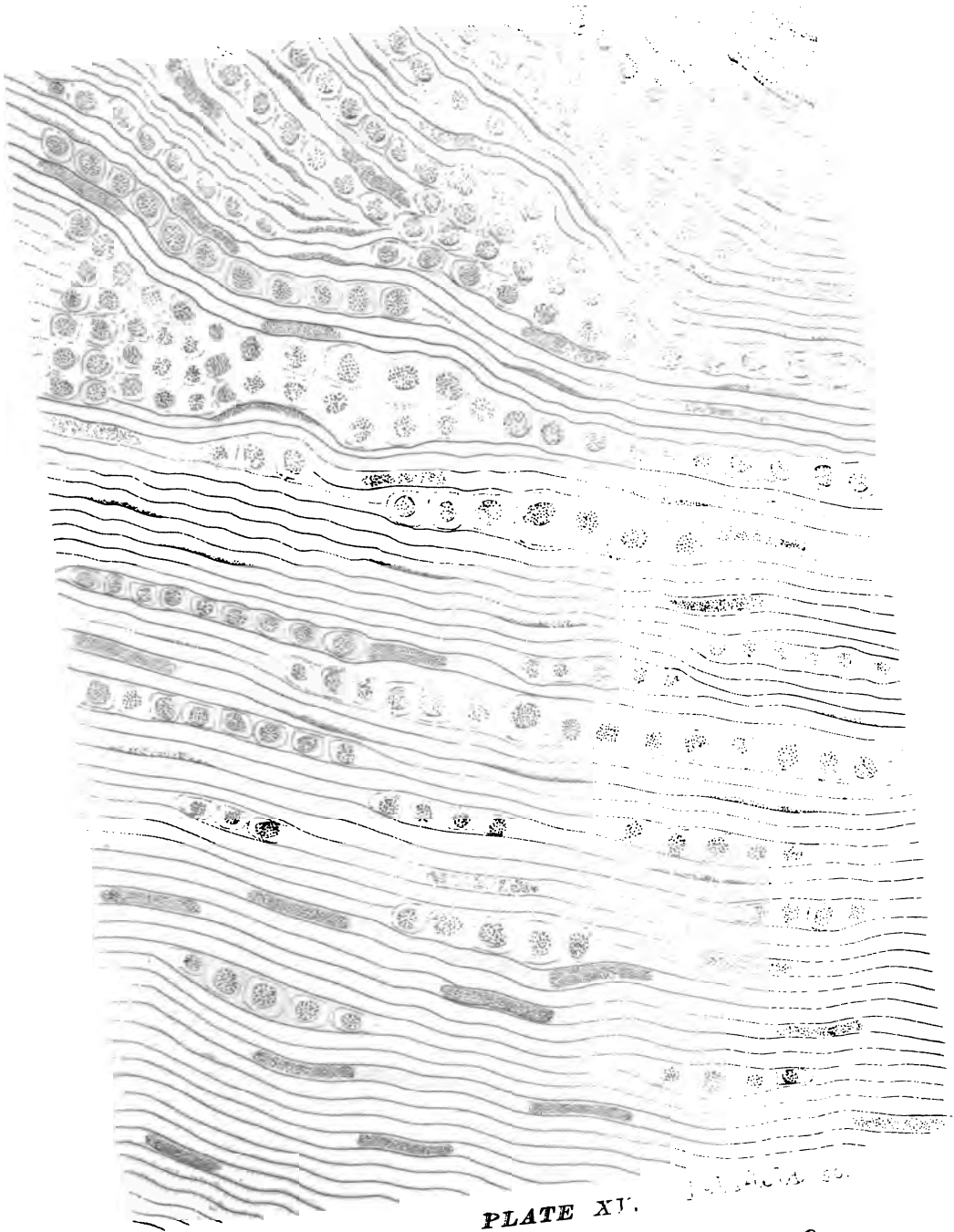


PLATE XV.

Vertical Section of the Human Pleura. Empyema.
Magnified 750 diameters.

recent cicatrix. The superficial layer of small cells is no longer seen, but is replaced by fusiform, stellate, and polygonal cells imbedded in an abundant fibrillated basement substance. If an empyema becomes gangrenous, the pleural cavity contains foul gases, the purulent serum is dirty and stinking, and swarms with bacteria. The fibrine coating the pleura is green or brown, and is infiltrated with bacteria. Sometimes smaller or larger portions of the pleura itself are destroyed. In such cases the destruction of tissue is not effected by suppuration, but by a molecular death and disintegration.

There are cases, however, in which the whole thickness of the pleura does suppurate and soften; the same process extends to the fasciæ, the muscles and the skin, and the pus is discharged externally. By modifications of the same process, the pus may be discharged through the bronchi, or perforate into the peritoneal cavity or into some of the abdominal viscera.

It is evident, therefore, that the regular course of an empyema is to produce very profound changes in the pleura. Its inner layers are converted into granulation tissue, its deeper layers into thick, dense, fibrous tissue. The pleura after such changes can never return to its normal condition. The utmost that can be expected is that the granulation tissue should be converted into cicatricial connective tissue, and that the formation of purulent serum should cease.

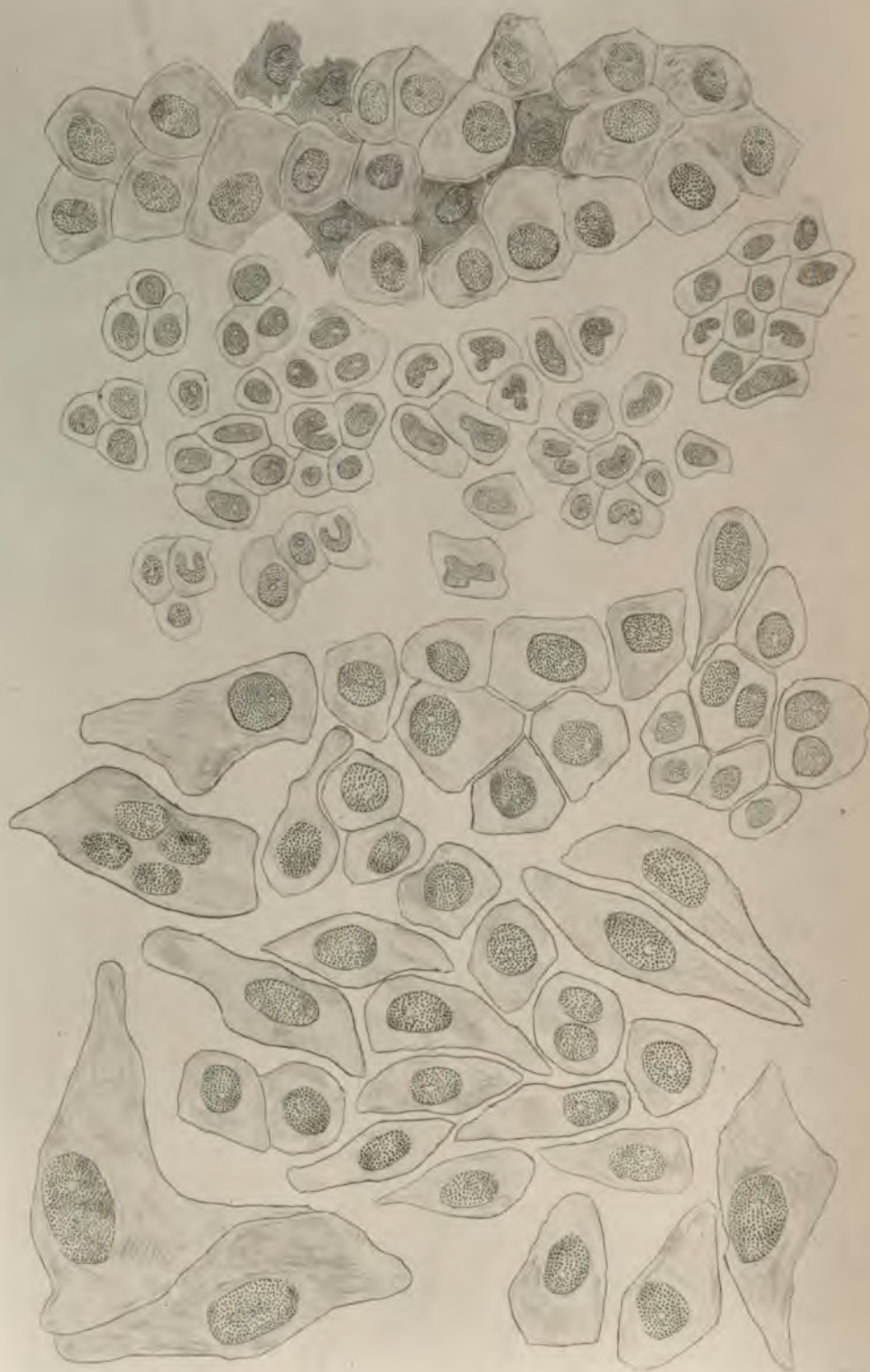


PLATE XVI.

*Endothelial Cells in Hydrothorax.
Magnified 750 diameters.*

HYDROTHORAX.

THE term hydrothorax is usually applied to dropsical accumulations of serum in the pleural cavities, in contradistinction to pleurisy with effusion. The ordinary causes of such dropsical effusions are valvular disease of the heart, chronic Bright's disease of the kidneys, and cirrhosis of the liver.

Although this condition of the pleura is not usually considered as an inflammatory condition, and although the serum is not of inflammatory origin, yet there are certain changes in the endothelium which do seem to be of an inflammatory nature.

If we examine the thorax of a patient who has died with general dropsy, we find either one or both pleural cavities partly filled with serum. This serum is usually clear at first, but, after exposure to the air, flocculi of fibrine may coagulate in it. The surface of the costal pleura is usually clean and smooth, and of nearly normal appearance. Sometimes, however, it is coated with a little fibrine, and sometimes it has a somewhat opaque and sodden appearance; or, if the dropsy is due to heart disease, the blood-vessels of the pleura may be dilated. The endothelium of the pleura, however, is as a rule much altered. The alteration consists in a change of the old endothelial cells, and the production of new ones beneath the old. Some of the old cell-bodies become irregularly shrunken and more opaque, so that they look like branching cells; other cells become hypertrophied and misshapen. If these old cells are brushed off, we find beneath them patches of small, polygonal, nucleated cells, arranged side by side, and having all the appearance of new endothelium. In some places the old endothelium has entirely disappeared, and the pleura is coated over a considerable area with the new cells. The fixed connective-tissue cells beneath the endothelium may also be somewhat increased in size and number.

Plate XVI. shows the appearance of the old and new endothelium—the old cells large and misshapen; the new cells small, but of regular shape.

The precise way in which the new endothelium is formed is not evident. It would seem that it must be developed either from the old en-

dothelium, or from the superficial connective-tissue cells, or from emigrated white blood-globules. I must confess, however, that I have not seen any positive proof of either of these modes of origin.

CHRONIC PLEURISY WITH ADHESIONS.

I employ this name, not to designate all those cases in which we find after death old pleuritic adhesions, either with or without a previous history of pleurisy, but to group together a set of cases which have a characteristic clinical history, as well as anatomical lesions. I can perhaps give the best idea of my meaning by a short history of a case.

A gentleman of rather slender physique, and disposed to dyspepsia and other smaller ailments, was actively engaged in business till he was sixty years old. He then retired from affairs and settled in the country. After about eighteen months he began to be troubled with a slight cough, with dyspnoea on exertion, and lost flesh and strength. He became alarmed about himself and consulted a number of physicians. The prevailing opinion was that he was suffering from pulmonary phthisis, and he was treated accordingly. He continued, however, to grow worse; the dyspnoea increased and was aggravated by irregular action of the heart. The cough was very troublesome, and was accompanied with scanty muco-purulent sputa. The loss of strength and flesh reached such a degree that he was confined to his bed. So he gradually wasted and died. At the autopsy there were no lesions found except of the thorax. The lungs were adherent to the wall of the thorax over their entire surfaces. Neither the pulmonary nor the costal pleuræ were much thickened. The adhesions were all formed of connective tissue; none of them were very dense, and some were extremely thin and delicate. In the superficial portions of the lungs were a few patches of interstitial fibrous tissue, but none of any size. The larger bronchi were trabeculated and coated with a little muco-pus. Except for a moderate degree of senile emphysema, the lungs were otherwise normal. The heart was pulled upward and to the right, apparently by the pleuritic adhesions.

Such cases as these come under the notice of every clinical observer.

They are all characterized by essentially the same symptoms: gradual loss of strength and emaciation, cough and dyspnœa, no acute invasion of the disease, but a slow and steady progress.

It is not easy to determine the exact pathology of these cases; they seldom die until the disease has existed a long time and the adhesions are already extensive. As a rule, both lungs are involved in about the same degree. The complicating bronchitis varies in its degree in different cases; in some being quite slight, in others sufficient to coat the bronchi with muco-pus.

Interstitial pneumonia is also a regular, complicating lesion, but it also is found in different degrees in different patients. In some cases there are large patches and bands of new fibrous tissue; in others it is only to be discovered by close examination. In all cases the new fibrous tissue begins just beneath the pulmonary pleura, and extends from thence inward into the lung.

Some of the pleuritic adhesions are quite thick, others so thin as to form mere films. Some are covered on both sides with a regular layer of endothelium, the basement substance is distinctly fibrillated, the fixed connective-tissue cells are not large or numerous, the blood-vessels are only in moderate numbers; these seem to be the oldest adhesions.

Other adhesions are also covered with endothelium, but the endothelial cells are of more irregular shape; the fixed connective-tissue cells are larger and much more numerous; the blood-vessels are also more abundant, and there are groups of pus-cells along their course; these adhesions seem to be younger, and still growing.

The thinnest adhesions are not covered with a continuous layer of endothelium, but with irregular patches; the fixed connective-tissue cells are numerous, large, with long, branching processes; the basement substance is not fibrillated, but more homogeneous. These seem to be the youngest adhesions.

The costal pleura between the adhesions does not present any marked change to the naked eye, except at those places where the adhesions are very close together. At such places the costal pleura and the adhesions form together a sort of loose, spongy tissue.

The pleura between the adhesions, although it presents no gross changes, is nevertheless altered. The endothelial cells are increased in size and number. There are giant-cells containing a number of nuclei, and in places the cells are heaped one upon another. The fixed connective-tissue cells are also increased in size and number. The blood-vessels are numerous and may project above the surface of the pleura; they are accompanied by a thick coating of connective-tissue cells and by groups of pus-cells. There are also bands of fibrillated tissue stretching across the surface of the pleura, looking like the commencement of adhesions. In Plate XVII. some of these changes are seen, especially the altered endothelium; the drawing represents the surface of the costal pleura between the adhesions.

We may, therefore, conjecture that in this variety of pleurisy the morbid changes begin in the endothelium and connective tissue, without any previous production of fibrine, and that the adhesions are formed by a direct outgrowth from the costal and pulmonary pleura.

THE PLEURISY OF CHRONIC PHTHISIS.

In the course of chronic pulmonary phthisis any one of the different varieties of pleurisy may be, and usually one of them is, developed; but there is one variety of pleurisy which occurs with chronic phthisis, and is never seen except with this disease.

It is perhaps more apt to occur with that form of phthisis in which the air-cells are filled with solid matter, so that the lung is large, does not contract and fills the pleural cavity almost completely.

Both the costal and pulmonary pleura are thickened, but are only adherent in places. The free surface of the pleura has a peculiar, roughened, lustreless appearance. If we make a vertical section through the thickened pleura, we find that its surface is covered with a thick layer composed of fibrine and of new tissue. If we tear off successive planes of tissue from the surface of the pleura, the same structure is evident. The peculiarity of the inflammation is this: that the production of fibrine and of new tissue seem to go on simultaneously. There is not, as in ordinary pleurisy, first a production of fibrine and then a

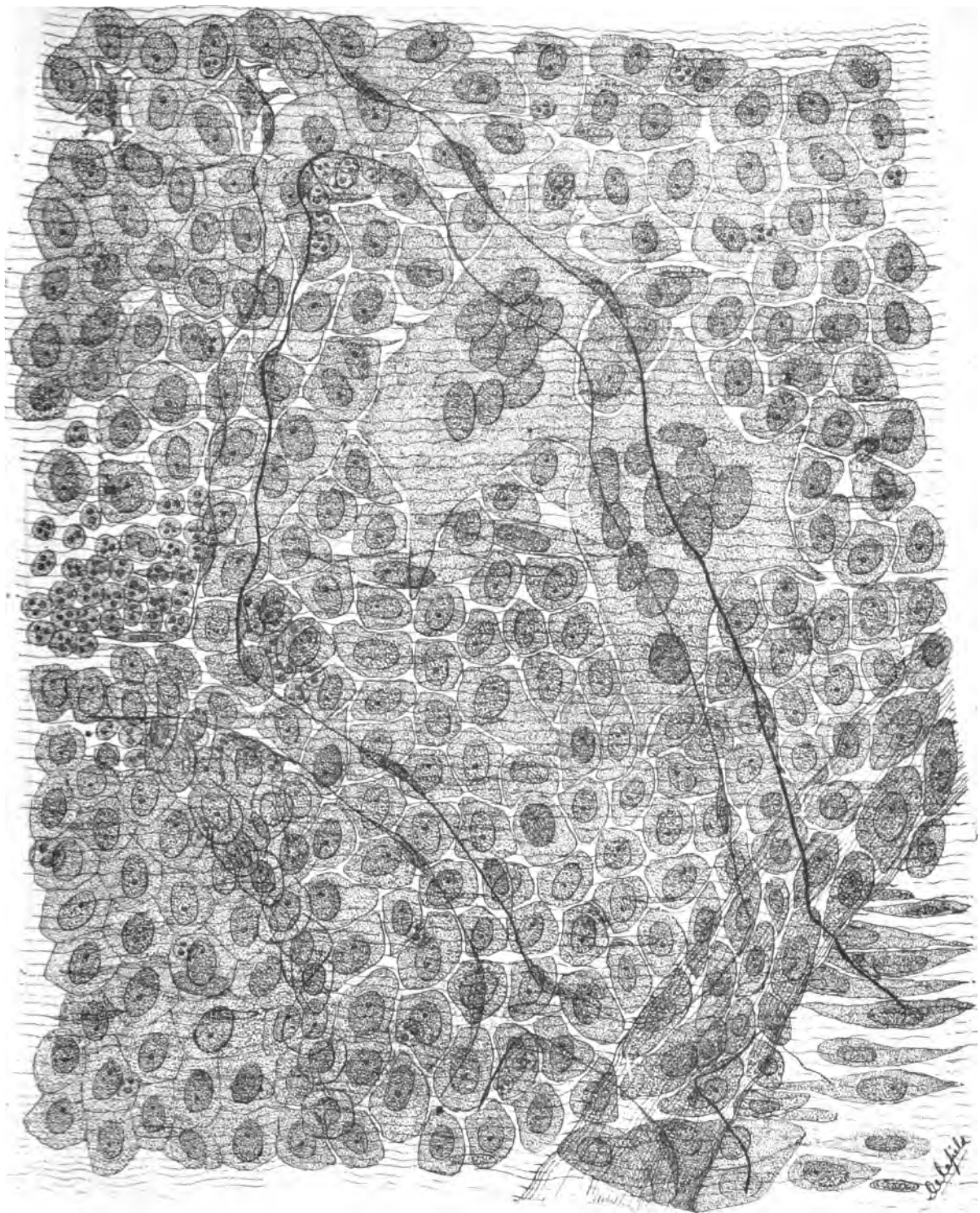
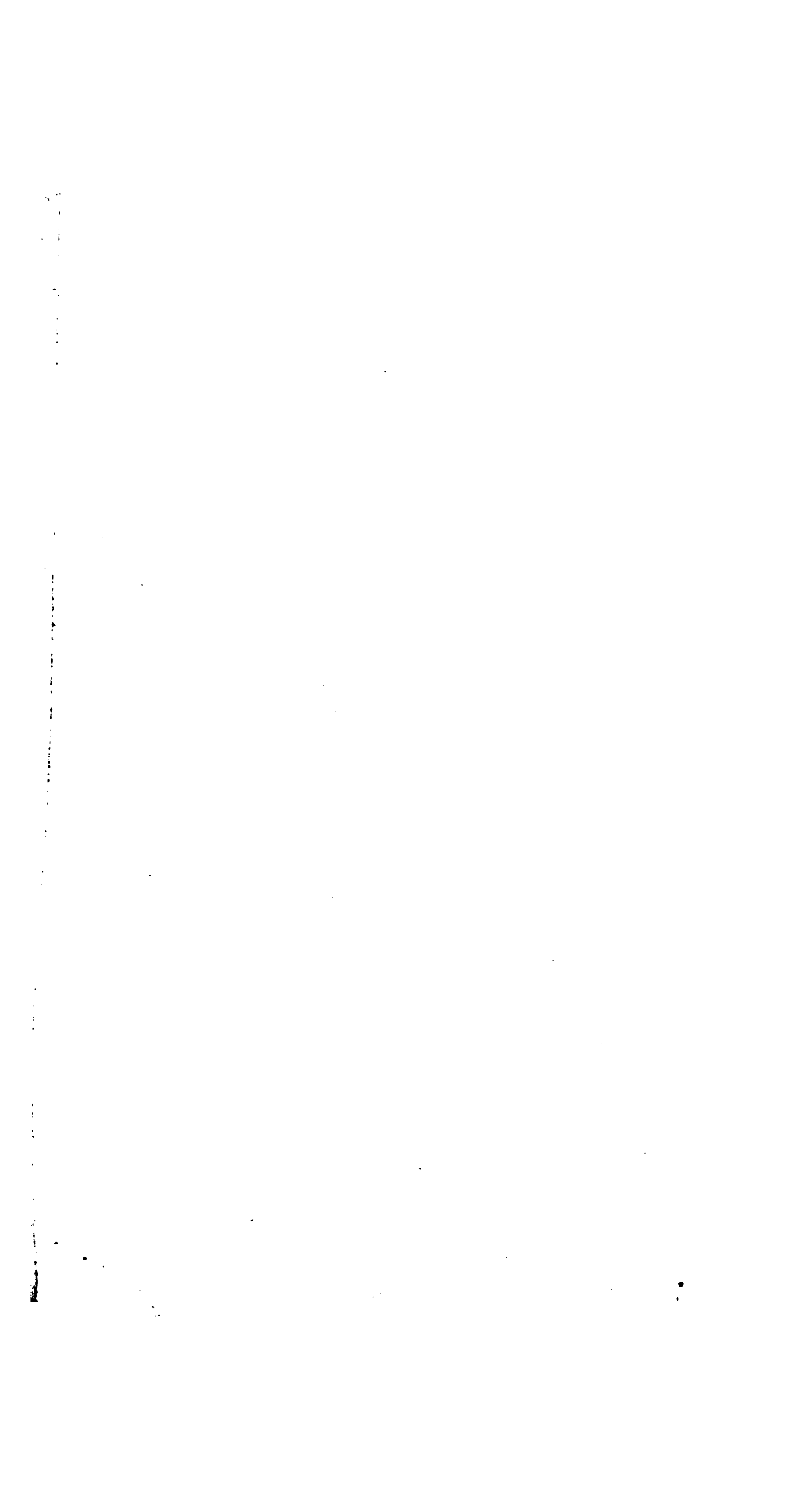


PLATE XVIII.

Conium maculatum
Conium maculatum



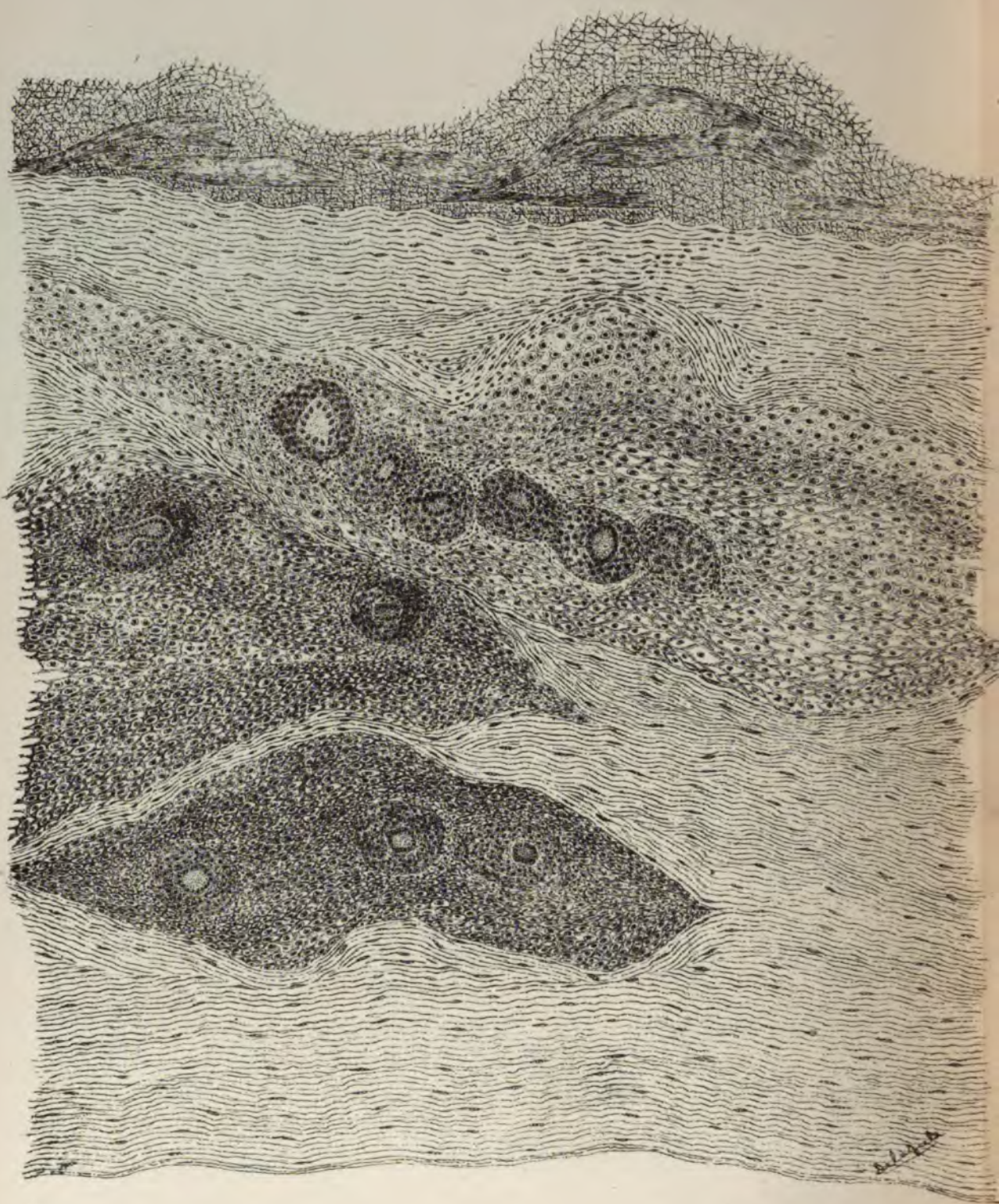


PLATE XVIII.

*Vertical section of the Pleura. Tubercular Pleurisy.
magnified 90 diameters.*

formation of new tissue and a disappearance of the fibrine. But these two processes are repeated over and over again, so that the layer on the surface of the pleura gives us an inextricable confusion of fibrine, of blood-vessels, and of new connective tissue in different stages of growth.

This form of pleurisy has also a clinical interest. The roughened surfaces of the pleura are well adapted to give a friction sound, and it is a question how many of the subcrepitant râles that we hear in such cases of phthisis are produced in this way.

TUBERCULAR PLEURISY.

Tubercular inflammation of the pleura occurs in the course of chronic pulmonary phthisis, as one of the lesions of acute general tuberculosis and as a local inflammation.

The appearance of the affected pleura varies with the amount and arrangement of the tubercles, and with the character of the other inflammatory lesions which accompany the tubercular inflammation.

The tubercle is usually arranged either in the form of small, circumscribed nodules of white, gray, or yellow color, miliary tubercles; or of larger, yellow, flat masses. It may be situated either on the surface of the pleura, or deep in its tissue. If we examine more closely the small miliary tubercles, we find that in some cases they have undergone cheesy degeneration and consist of nothing but granular matter; in other cases they are largely composed of small, round cells which cover over and obscure the tubercle proper; while in still others the tubercular foci are so small as to be hardly visible to the naked eye, but are not degenerated or obscured by other products of inflammation. When this latter condition prevails, we find the tubercle in its characteristic forms of tubercle-granula and diffuse tubercle. The granula are formed of polygonal, nucleated cells and giant-cells arranged in a reticulated basement substance, while the diffuse tubercle is composed of the same tissue spread out diffusely. There is at the same time a multiplication of the fixed connective-tissue cells of the pleura. Plate XVIII. represents a vertical section of the pleura, including its entire thickness, and showing the arrangements of the tubercle-granula and diffuse tubercle.

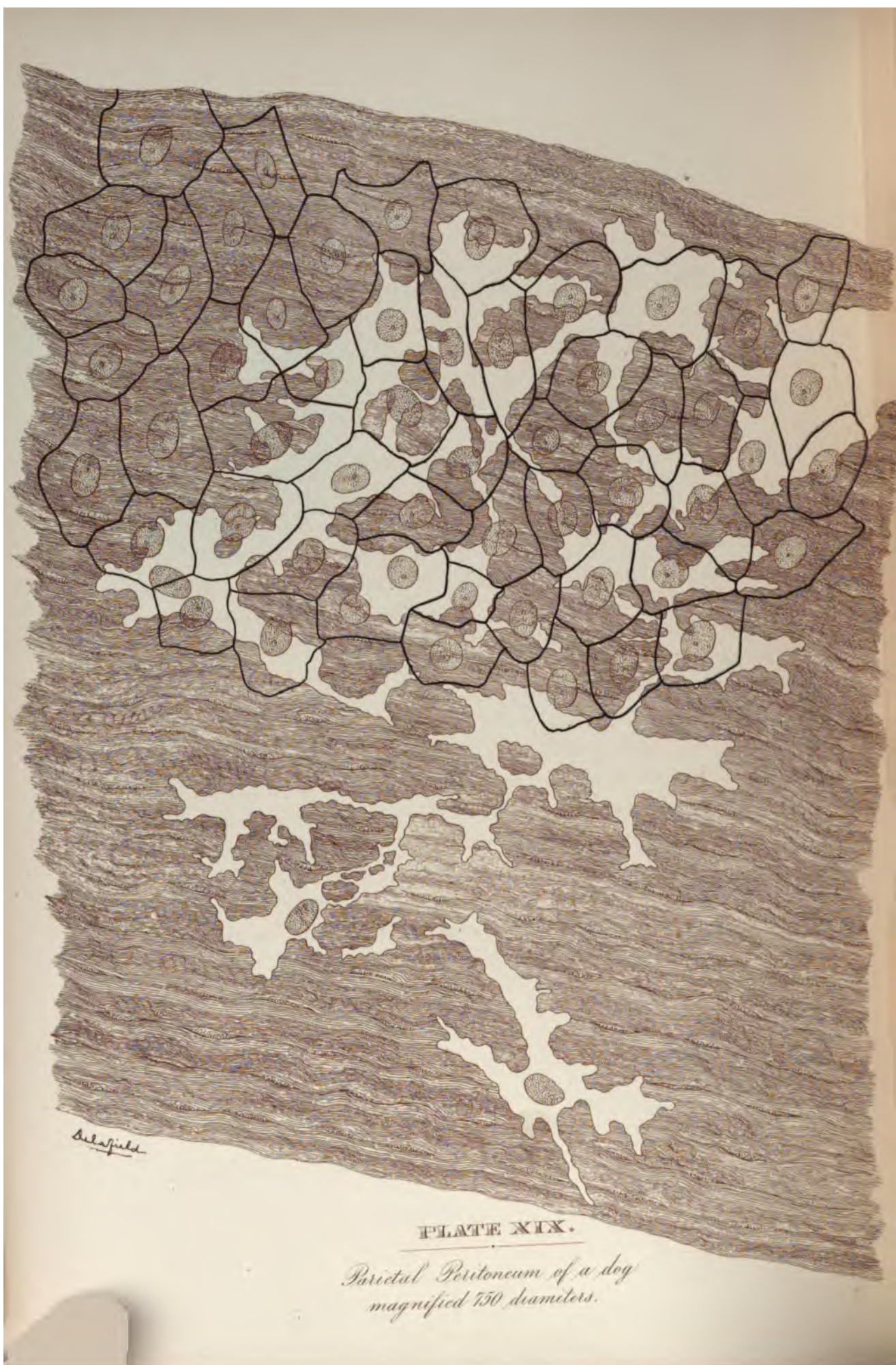


PLATE XIX.

*Parietal Peritoneum of a dog
magnified 750 diameters.*

THE PERITONEUM.

THE abdomen, like the thorax, forms a cavity containing viscera. The inner surface of this cavity and the surfaces of the viscera are covered with connective-tissue membranes, and folds of similar membranes hold the viscera in place. All these membranes are considered by anatomists to form together a closed serous sac called the peritoneum. The membrane lining the wall of the abdomen is termed the parietal peritoneum; and that covering the viscera, the visceral peritoneum. The membranes which hold in place the small intestines—the cæcum, the transverse colon, the sigmoid flexure, and the rectum—are called respectively the mesentery, the meso-cæcum, the transverse and sigmoid mesocolon, and the meso-rectum. Certain folds of membrane which are spread out over the viscera are called the great omentum, the small omentum, and the gastro-splenic omentum. Still other membranes, which hold in place the liver, spleen, uterus, and bladder, are called ligaments.

The parietal peritoneum resembles in its structure the costal pleura. Its free surface is covered with a single layer of flat, polygonal, nucleated cells—the endothelium. Beneath these cells are successive planes of connective tissue extending down to the muscles and fasciæ. These planes are formed of a fibrillated basement substance, reinforced by elastic fibres, and of branching cells like those of the costal pleura.

Imbedded in the connective tissue are lymphatic spaces and vessels; the spaces are numerous, but the vessels are not nearly as large or abundant as those of the costal pleura. The blood-vessels are very numerous, and break up into a capillary plexus with close meshes. See Plate XIX., which represents the endothelium, basement substance, connective-tissue cells, and lymphatic spaces.

The mesentery has two free surfaces, each covered by endothelium. In it are the large blood-vessels and lymphatic trunks which pass to the intestines, besides a system of blood and lymphatic vessels belonging to itself.

The omentum in the human subject and in some animals consists of fibrillated connective tissue arranged in a mesh-work. The trabeculæ of this mesh-work are completely covered by large endothelial cells. In the basement substance, beneath the endothelium, are branching cells, which in the normal omentum are not readily seen, but become swollen and plainer when inflamed. In the larger trabeculæ are blood and lymphatic vessels and fat. Plate XX. represents part of the normal human omentum.

Klein has called attention to the fact that in the omenta of different animals are to be found small nodules, and that these nodules are not all of the same structure. Some, he says, are formed of germinating endothelium; some of branched cells; some of adenoid tissue; some of blood-vessels and branched cells. In the human omentum such nodules are also to be seen, especially if the individual has suffered from heart disease or cirrhosis of the liver. They are formed of an accumulation of cells resembling the endothelium, but smaller and with thicker cell-bodies; or of accumulations of branched cells, with a network of capillary vessels. It is also not uncommon to find from two to ten nuclei of endothelial cells close together.

THE INFLAMMATIONS OF THE PERITONEUM.

Although the peritoneum resembles the pleura very closely in its structure, yet the inflammations which invade these two membranes differ widely in their anatomy and clinical history. One reason for this

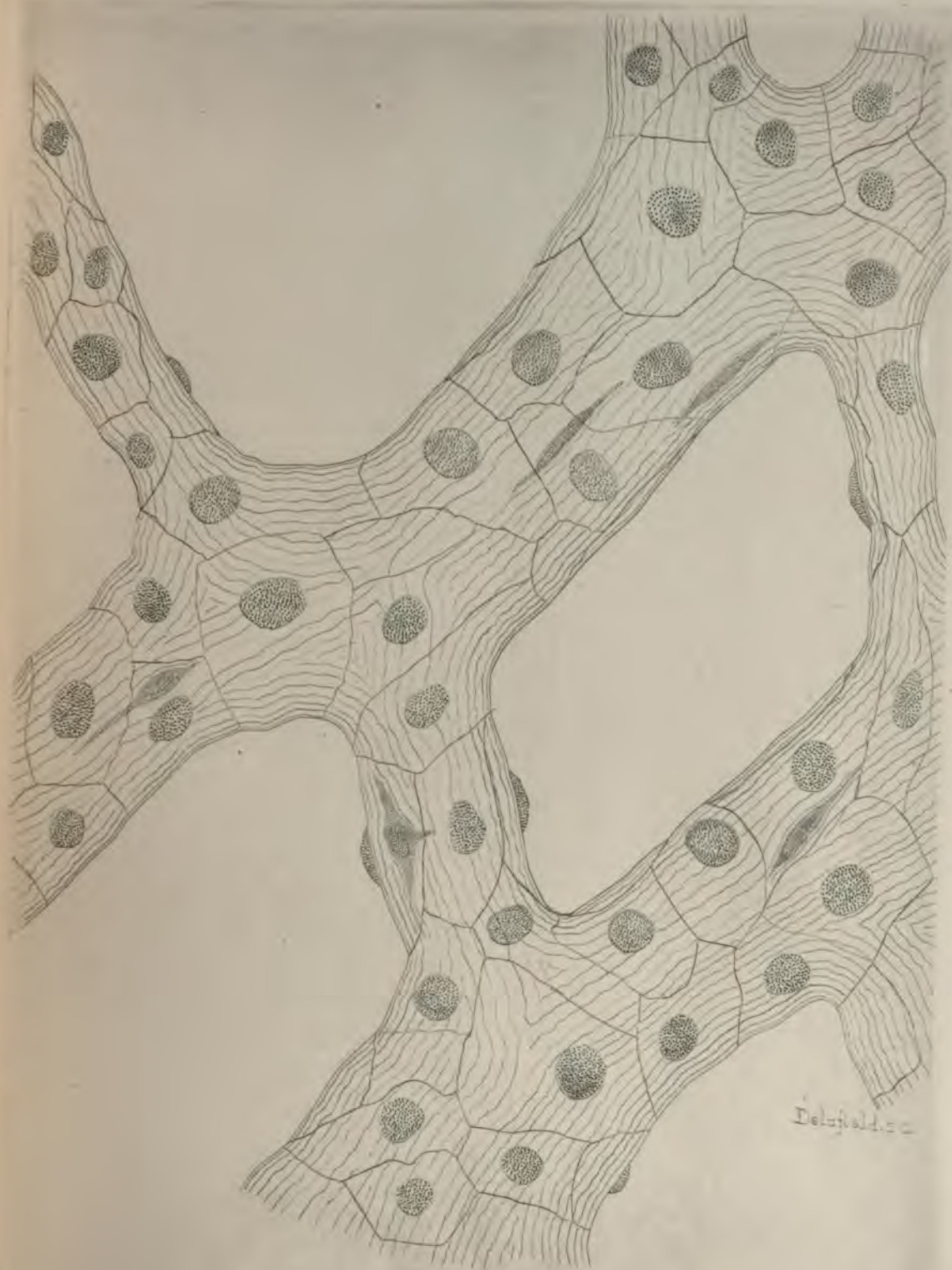


PLATE XX.

*Normal Human Omentum.
Magnified diameters.*





PLATE XVI

*Cellular Pulp of human
enlarged 250 times*

alveolar

difference is that peritonitis is a much more serious lesion than pleurisy, and causes death in a much shorter time. Pleurisy is not necessarily a fatal, or even a severe disease; peritonitis produces a very marked change in the general condition of the patient, and is very often fatal. A well-marked acute peritonitis usually destroys life within from two to fourteen days.

We cannot, therefore, classify peritonitis as we do pleurisy, according to the anatomy of the inflammation, but rather we follow its clinical history and speak of acute and chronic peritonitis; or its extent, and speak of local and general peritonitis.

I. Acute Peritonitis.

We can distinguish two anatomical varieties of acute peritonitis. There may be no change except in the endothelium; or there may be changes in the endothelium and fixed connective-tissue cells, with production of fibrine, serum, and pus. This last is the most frequent form of acute peritonitis.

1. The variety of acute peritonitis in which the changes are confined to the endothelium we may call, for convenience, *Cellular Peritonitis*. This form of peritonitis may be produced by any irritant which does not act too energetically. It can be excited in dogs by injections of very small quantities of a solution of chloride of zinc. In the human subject we find it with perityphlitis, with abscesses shut in in any part of the peritoneum, and in cases of puerperal fever which die within forty-eight hours after the development of symptoms.

If the autopsy is made within a few hours after death, we find the entire peritoneum of a bright red color from the congestion of the blood-vessels; but this is all—there is no fibrine, no serum, no pus. If, however, we examine the peritoneum with the microscope, we find a well-marked change in the endothelium, especially that of the omentum. The cells are increased in size and number, and present such an appearance as is seen in Plates XXI. and XXII. Plate XXI. shows the human omentum in a case of perityphlitis; Plate XXII. the omentum of the dog on the fourth day after an injection of chloride of zinc.

The following case may serve as an example of this form of peritonitis:

A boy, seventeen years old, nine days before his death was suddenly seized with severe pains in the right lumbar and iliac regions. He went home, and in the evening had a chill and felt ill. The next day he had two chills, vomited everything he ate, and was delirious. The third day he managed to walk to a dispensary, although still suffering and feeling very ill. His bowels were moved on that day, and continued to be regular until two days before he died. On the fourth day his pulse was 100, temperature 104° ; there was tenderness and swelling in the right iliac fossa. On the fifth day his pulse was 104-112, temperature $101-100^{\circ}$. On the sixth day the pulse was 106, temperature $99\frac{1}{2}^{\circ}$; there was more general tenderness and pain over the abdomen, and the boy looked worse. On the seventh day his pulse was 124-140, temperature $99-104^{\circ}$; he was sweating profusely, and was worse. On the eighth day his pulse was 144, temperature 103° ; he was delirious, his face drawn and anxious, his abdomen tympanitic, his urine was passed involuntarily, and on the ninth day he died. At the autopsy there was found general congestion of the entire peritoneum, but no fibrine, pus, or serum. The vermiform appendix was behind and adherent to the posterior wall of the cæcum. In the appendix was a large fecal concretion, and around this the wall of the appendix was infiltrated with pus; around this was a small collection of pus completely shut in.

It seems evident that this form of peritonitis, although not affording marked gross lesions, is yet capable of giving clinical symptoms. It is worthy of special notice as an example of inflammation producing new cells without the presence of fibrine, serum, or pus. We will see later that it is also of importance in reference to the pathology of tubercle.

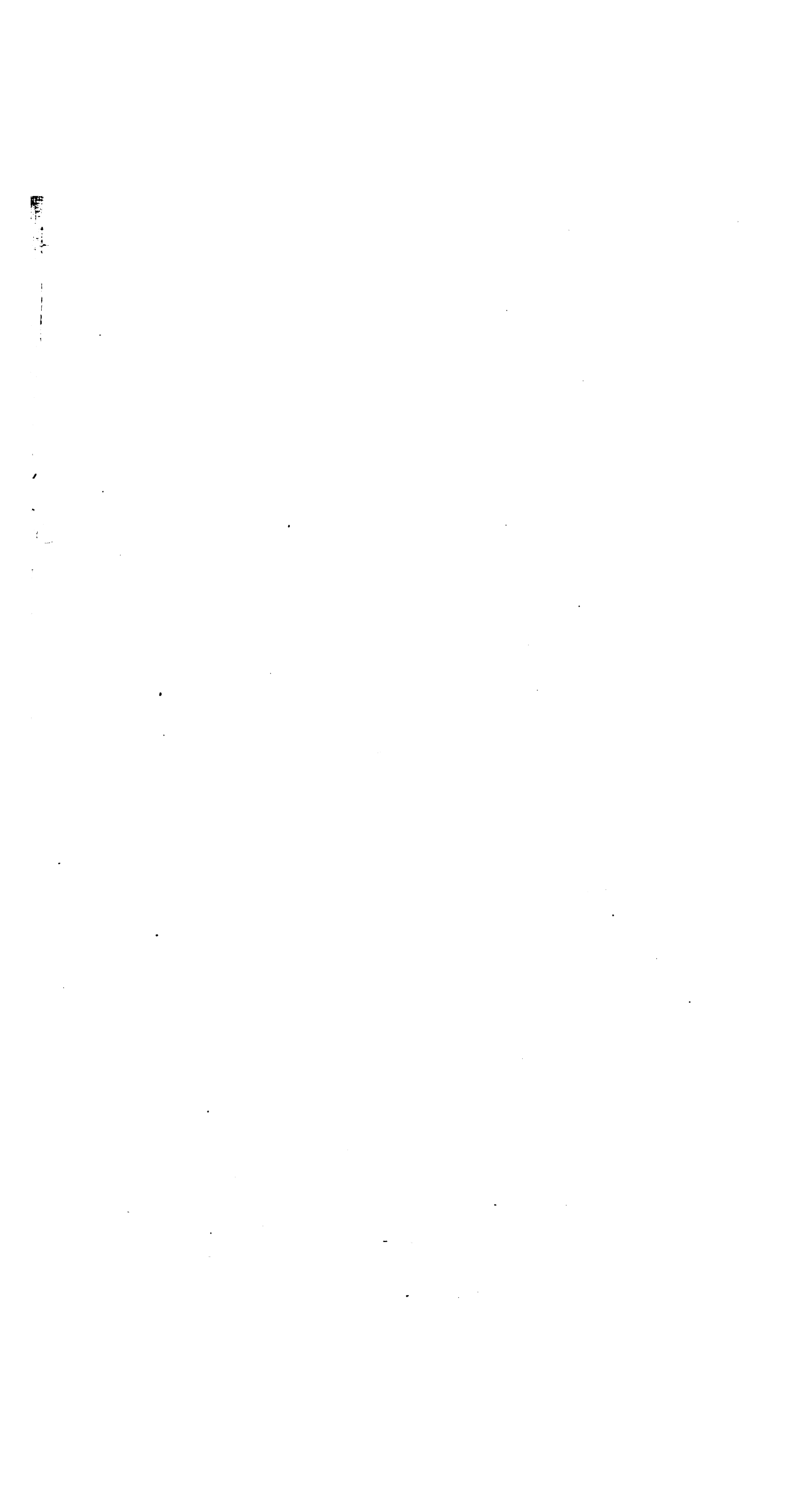
2. The ordinary form of acute peritonitis is attended with changes in the endothelium and fixed connective-tissue cells, and with the production of serum, fibrine, and pus.

Such a peritonitis may be produced by wounds or contusions of the abdomen; by ulcers, inflammations, new growths, and incarcerations and intussusceptions of the stomach and intestines; by lesions of the uterus, ovaries, and Fallopian tubes; by ruptures, inflammation and new growths of the bladder; by abscesses in the kidneys; by abscesses and hydatid cysts of the liver; by inflammation of the gall-bladder and biliary pas-



PLATE XXII.

*Cellular Peritonitis in a Dog; 15th day.
Magnified 750 diameters.*





del. & sculp.

PLATE XXIII.

*Cmentum of Rabbit, inflamed for one hour, connective tissue cells
and pus cells. magnified 750 diameters.*

sages; by thrombosis of the portal vein; by inflammations and new growths of the spleen and pancreas; by inflammations and new growths of the lymphatic glands and retro-peritoneal connective tissue; by abscesses in the abdominal wall; by perforation of an empyema; by inflammations of the vertebræ, ribs, and pelvic bones. It also occurs with septicæmia, with puerperal fever, with the acute exanthemata, with scurvy, with Bright's disease, and as an idiopathic lesion.

In studying peritonitis we must first observe the changes in the mesentery of the frog, following the now famous experiments of Cohnheim.

If we open the side of the abdomen of a curarized frog, pull out a loop of intestine and spread it over a cork ring, we have the mesentery so arranged that we can look at it directly with the microscope. The contact with the air is sufficient to excite a peritonitis.

The first change is that the arteries, then the veins, and then the capillaries become dilated, the current of blood within them is slower, and in some of the capillaries is stationary. Next we observe that there is an accumulation of white globules in the peripheral portion of the blood-current, and these white globules remain almost stationary, lining the walls of the vessels while the central column of red globules flows on uninterruptedly; this change is most marked in the veins. Next we see, on the outside surface of the walls of the veins, little knobs and projections making their appearance. These little knobs send out fine processes in every direction, they become larger and larger, and finally there appear on the outside of the veins bodies identical in appearance with white blood-globules and pus-cells.

In the capillaries these changes are to be seen more distinctly. We can see a single white globule moving slowly in the blood-current, adhering to some point of the wall of the vessel, then a little prominence appearing on the outside of the wall of the vessel, then the white globules inside becoming smaller and the prominence outside becoming larger until the entire globule appears on the outside of the vessel. In the same way the red globules may be seen to emigrate.

After these changes have gone on for several hours, the veins are surrounded by a thick layer of white globules, and after a still longer

time the entire mesentery is coated with a layer of white globules entangled in coagulated fibrine. Such an experiment shows that in the peritonitis of frogs the pus-cells are emigrated white blood-globules.

If we inject a solution of chloride of zinc or of ammonia into the peritoneal cavity of a rabbit or dog, we usually find that, by the end of one or two hours, inflammatory changes are evident. There is a little serum in the peritoneal cavity, and little knobs and threads of fibrine can be seen on the surface of the peritoneum. The endothelium shows little or no changes, nor do the fixed connective-tissue cells. Pus-cells, however, are already present in moderate numbers in the stroma of all parts of the peritoneum just beneath the endothelium. In the dog the pus-cells have no connection with any other cells; in the rabbit they are sometimes so close to the branching cells (as seen in Plate XXIII.), that it is difficult to be certain of their relationship.

After the lapse of twenty-four hours the lesions are more marked: there is general congestion of the peritoneum, reddish serum in its cavity, and an appreciable coating of fibrine and pus on its surface.

Minute examination shows that two distinct sets of changes are going on at the same time: 1, a production of fibrine, serum, and pus; 2, a swelling and multiplication of the endothelial cells. If the inflammation is very intense, the pus and fibrine are most abundant; if the inflammation is milder, the changes in the endothelium are more marked.

There may be a considerable amount of pus produced and yet the layer of endothelium remain in place. But, in silver preparations, little black spots are seen along the edges of the cells, and at many points there are large, rounded openings between the cells, in some of which appear to be pus-globules. The pus-globules are seen in numbers beneath the layer of endothelium, and may infiltrate the entire thickness of the peritoneum. They seem to have no connection with the fixed connective-tissue cells, and the latter are only a little swollen. The blood-vessels contain a very large number of white globules. Plate XXIV. represents the parietal peritoneum of a dog, inflamed for forty-eight hours; the endothelium is in place, but there are large openings between some of the cells, and numbers of pus-globules can be

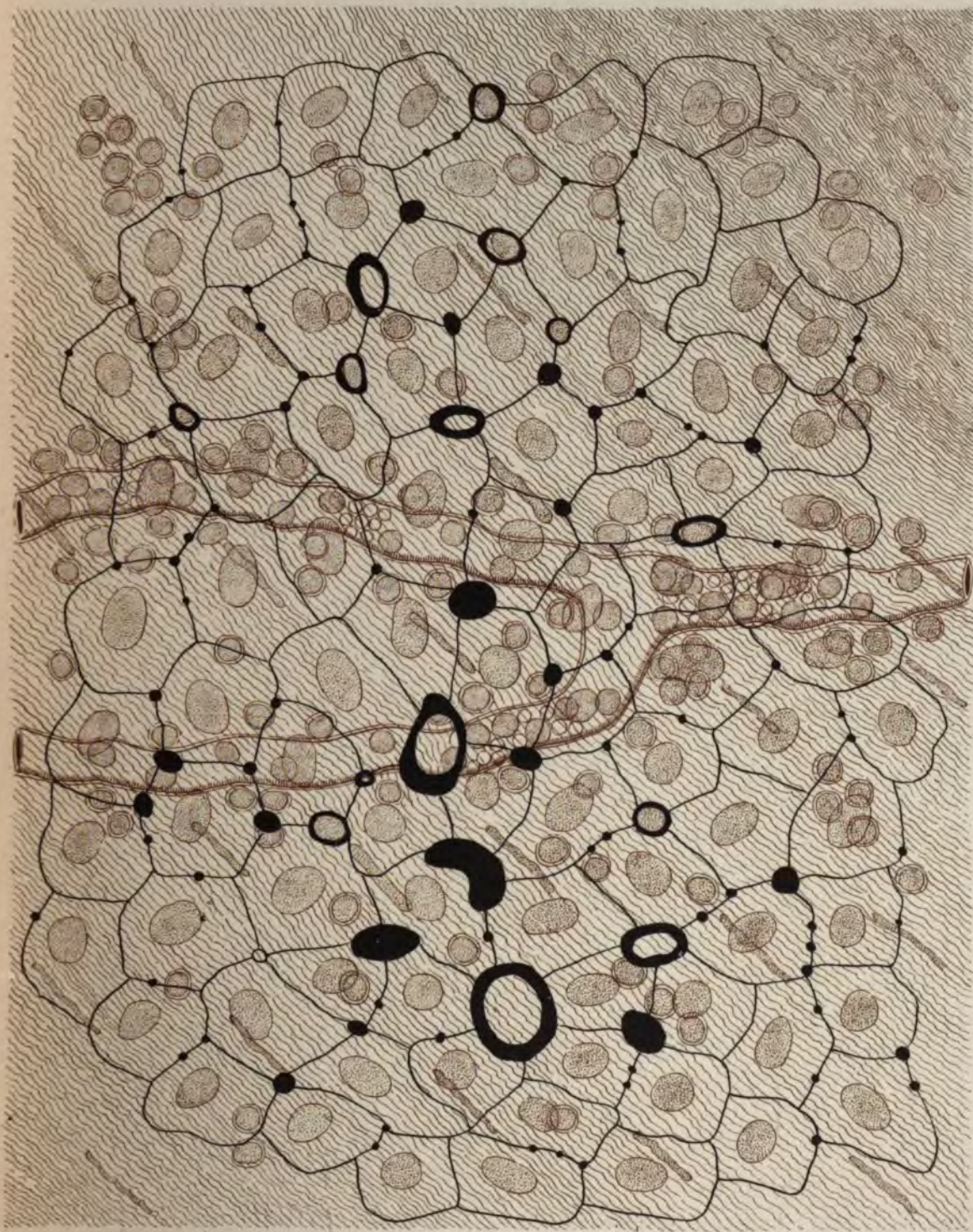
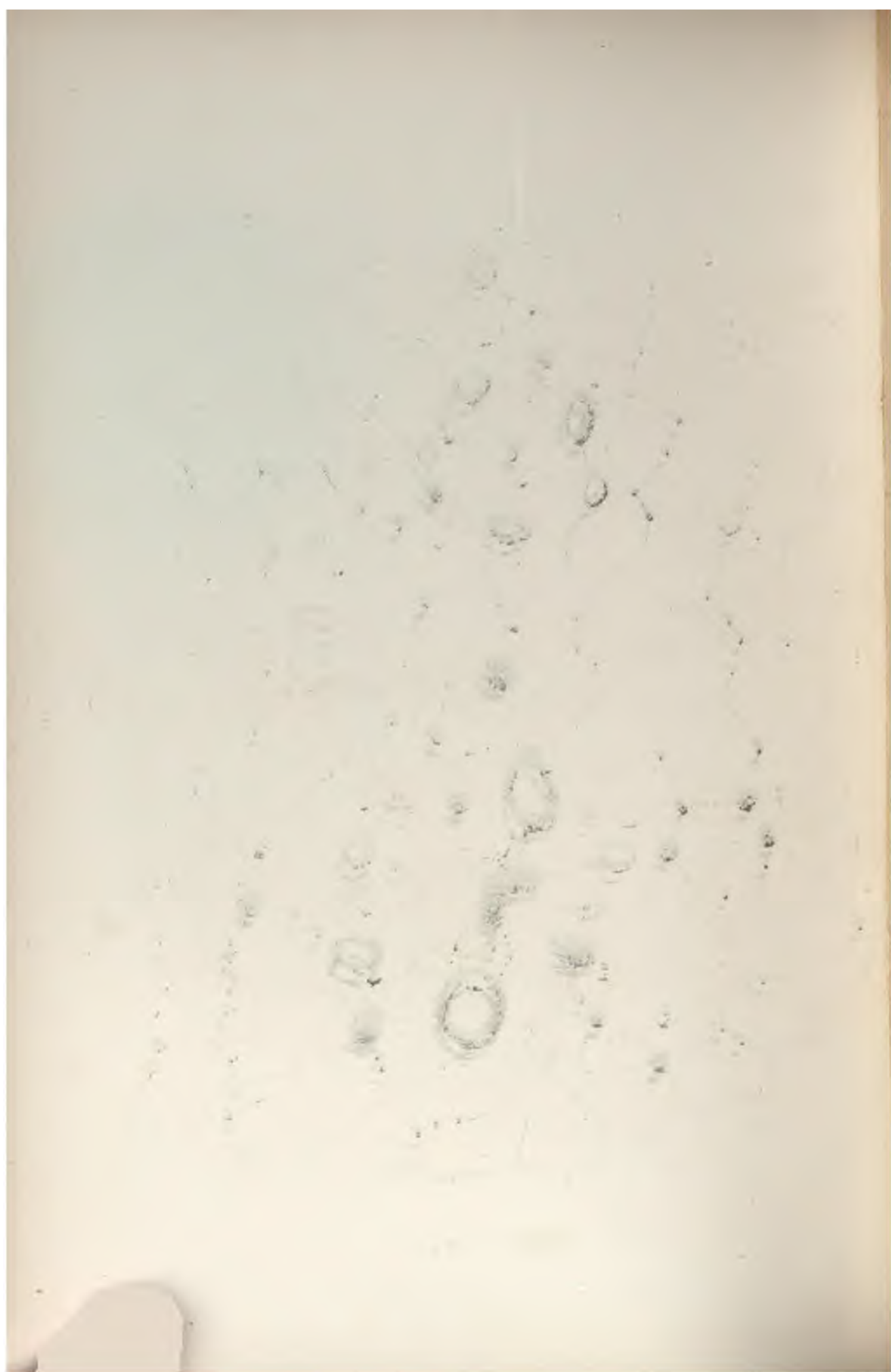
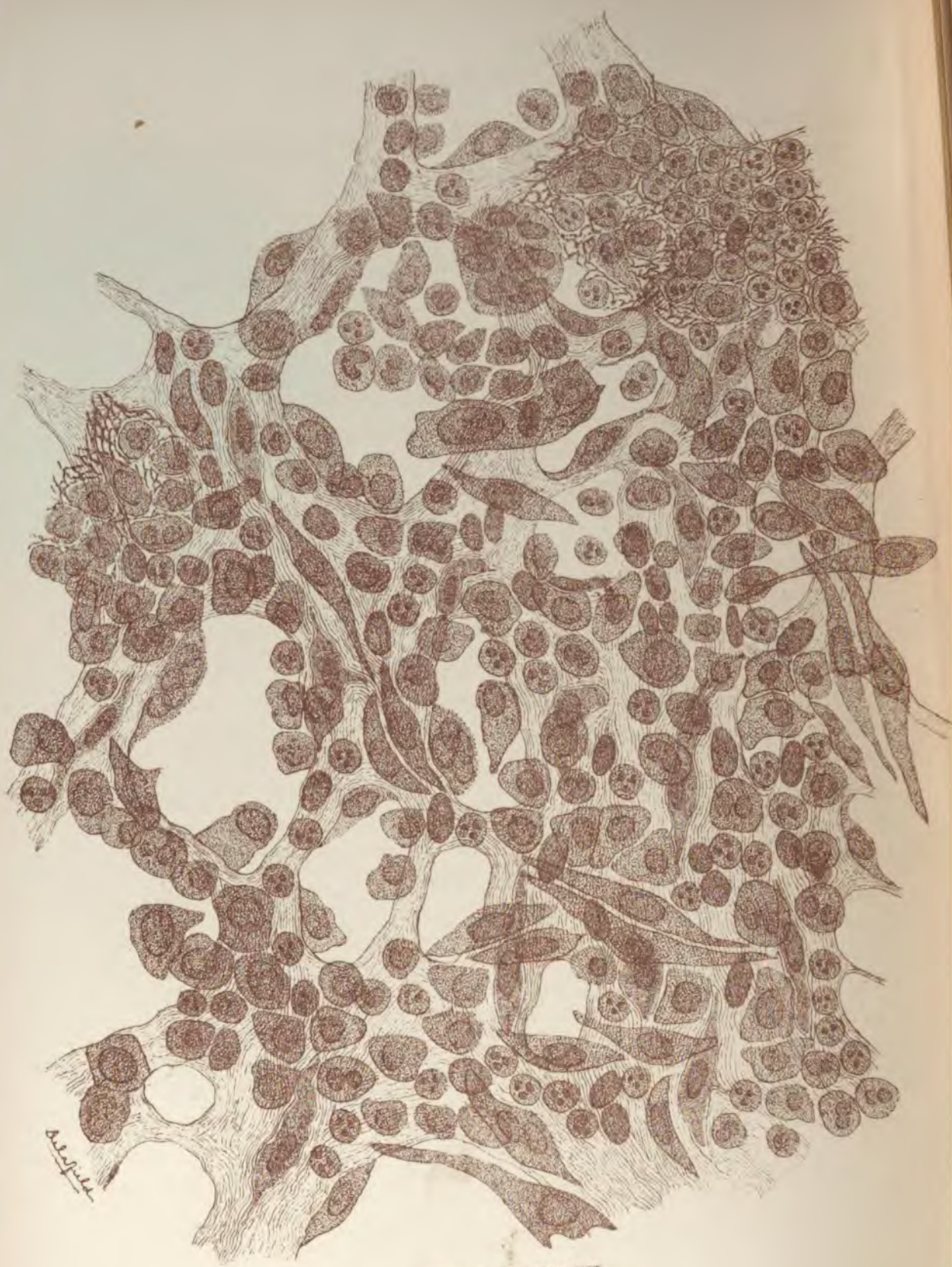


PLATE XXIV.

*Peritonitis in a dog for 48 hours,
magnified 250 diameters.*







Beesfield

PLATE XXV.

*Human Omentum, acute peritonitis,
magnified 750 diameters.*

seen beneath the endothelium, while some of the capillaries are filled with white globules.

If, however, the pus and fibrine are produced in large amounts, the endothelium falls off and leaves the surface of the peritoneum bare.

The swelling and multiplication of the endothelium give such pictures as are seen in Plates XXI. and XXII.

These two sets of changes go on in the dog up to the third day, the serum, fibrine, and pus increasing in amount, and the endothelial cells increasing in size and number. But few dogs survive the third day of a well-marked acute peritonitis.

In the human subject, if death takes place before the third day, both the gross and minute changes are the same as those seen in the dog. There is the same general congestion, the same production of pus, fibrine, and serum, the same desquamation and multiplication of the endothelium.

In many cases, however, of peritonitis, death occurs between the sixth and fourteenth days of the disease. The appearance of the peritoneum at this period of the inflammation is not always the same. The congestion of the vessels may persist, it may be very intense and accompanied with extravasations of blood, or it may be entirely absent. There may be a thin coating of fibrine and pus gluing together neighboring surfaces of peritoneum, or this layer may be thick and may shut in collections of pus. The lesions may be superficial or may infiltrate the subperitoneal tissue. There may be a small or a large quantity of purulent serum in the peritoneal cavity, and this serum may contain a few or a great many pus-globules, or the serum may be of a dirty brown, fetid character, filled with bacteria.

The microscopical appearances differ from those seen after forty-eight hours, chiefly in the larger amounts of inflammatory products and in the changes in the fixed connective-tissue cells. It has been stated already that during the first three days of a peritonitis the connective-tissue cells beneath the endothelium are but little changed; but by the seventh day these cells are markedly increased in size and number in all parts of the peritoneum. Plate XXV. shows the condition of the human omentum in a peritonitis of eight days' duration.

The rule is for peritonitis to prove fatal by the fourteenth day. Sometimes, however, the inflammation becomes chronic and may continue for a long time. Sometimes the patients recover and permanent adhesions are formed between different parts of the peritoneum; and these adhesions may shut in collections of pus. It seems probable that these adhesions are formed by the new cells derived from the endothelial and connective-tissue cells.

Whether recovery from peritonitis takes place without the formation of permanent adhesions, I have no personal knowledge, although it seems probable that this may take place.

Acute local peritonitis is often followed by the formation of permanent adhesions; this is especially the case in the peritoneum about the uterus, the liver, and the spleen. I have not been able to follow all the steps of such forms of peritonitis, but, so far as I can tell, the process is much the same as in acute pleurisy: first, the production of a layer of fibrine and a little pus on the surface of the peritoneum; then a growth of the superficial fixed connective-tissue cells to form permanent new tissue.

Acute general peritonitis differs, therefore, from acute pleurisy. In acute pleurisy, fibrine and serum are produced in large amounts, but not much pus; the endothelium has no active share in the inflammatory process; the superficial connective-tissue cells begin to multiply very soon, and from them are formed permanent new tissue. As the inflammation is not a fatal one, weeks and months may elapse before it has run its course.

In acute peritonitis there is usually a good deal of pus with the fibrine and serum; the endothelium does undergo marked changes; the connective-tissue cells are multiplied, but they do not go on to form permanent new tissue. The inflammation is a fatal one, and runs its course within a few days.



PLATE XXVI.

*Human Omentum, Chronic Cellular Peritonitis.
Magnified 750 diameters.*

II. Chronic Peritonitis.

Chronic peritonitis occurs in several anatomical forms, most of which can be conveniently grouped under the following heads:

1. Cellular peritonitis.
2. Peritonitis with adhesions.
3. Peritonitis with thickening of the peritoneum.
4. Peritonitis with adhesions, pus, fibrine, and serum.
5. Hemorrhagic peritonitis.

1. Cellular Peritonitis. This variety of peritonitis resembles very closely acute cellular peritonitis. There is no fibrine present and no pus, but there may be clear serum in the cavity of the peritoneum. To the naked eye there may be no changes visible; or we may see on the omentum minute semi-translucent granules, and on the parietal peritoneum small, rounded elevations. With the microscope, however, we find constant changes in the endothelium and fixed connective-tissue cells. These cells are everywhere increased in number and altered in shape; or, to speak more guardedly, the surface of the omentum is covered with cells which look as if they were derived from the endothelium and connective-tissue cells, although they differ from the normal shape of these. The new cells are for the most part polygonal cells of different sizes, with one or several nuclei, and giant-cells—large granular masses filled with nuclei. Although the new cells are produced over the entire surface of the peritoneum, yet as a rule they are more numerous in little patches scattered here and there. These little patches may be composed of only a few cells, or the cells may be heaped together in such numbers as to form nodules visible to the naked eye. There is never any stroma between the cells. Plate XXVI. represents the omentum from a case of chronic pulmonary phthisis, showing the production of new cells.

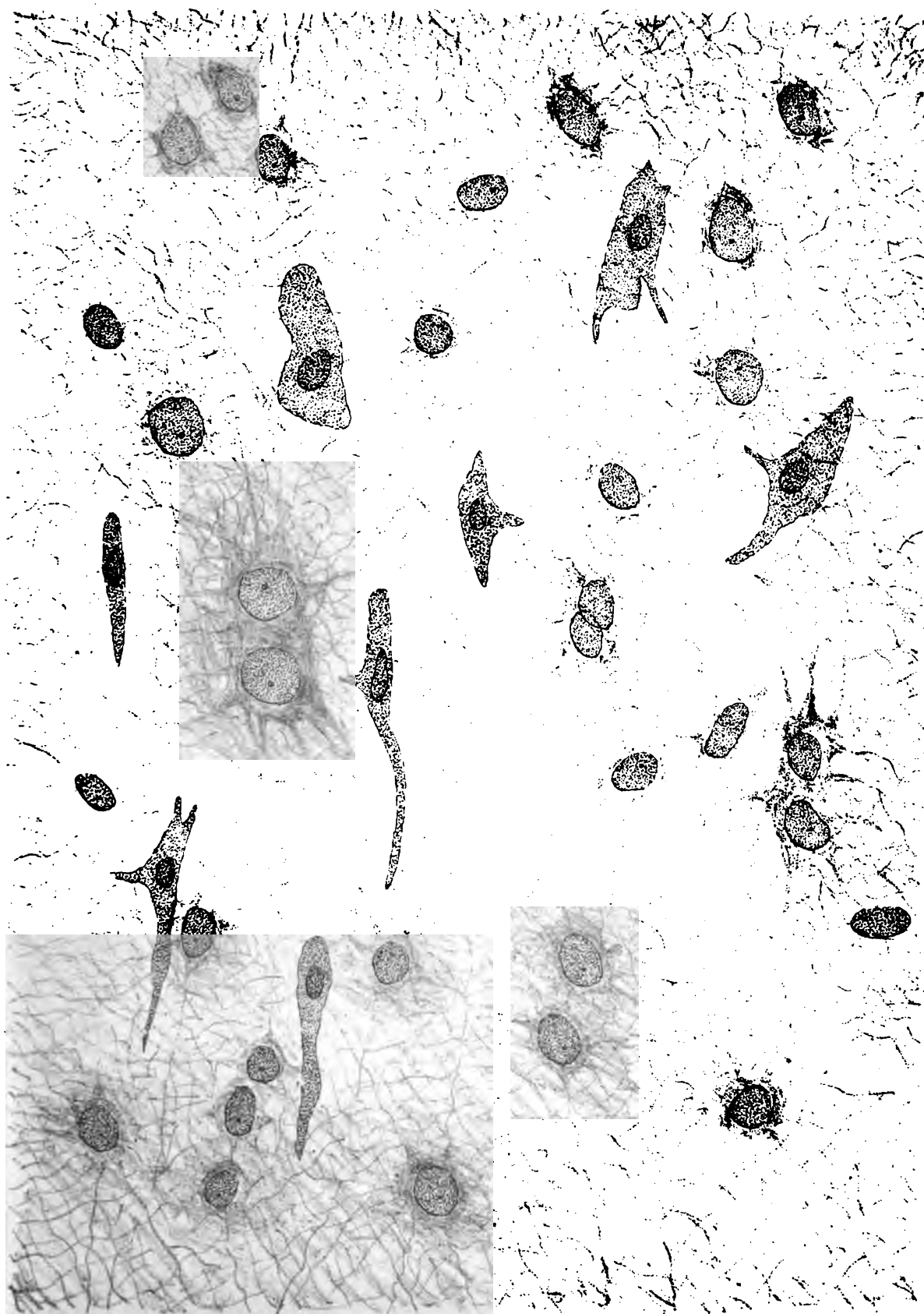
This form of peritonitis occurs most frequently with organic heart disease, with cirrhosis of the liver, with chronic pulmonary phthisis, and with acute general tuberculosis. With the two latter diseases the nodules produced by the accumulation of cells are often called tubercles—as it seems to me very improperly.

2. Peritonitis with Adhesions. There may be a formation of permanent adhesions, without the previous production of fibrine and pus. It is often, indeed, not easy to be certain whether old peritoneal adhesions are the result of an acute peritonitis, or whether they have been formed without any such preceding inflammation; in many cases we have to rely on the history of the patient to solve the problem.

There are, however, cases in which the manner of growth of such adhesions seems clear. If, either from perityphlitis or from any other cause, a collection of pus is shut in, in some part of the peritoneal cavity, we may find the rest of the peritoneum smooth and shining; no serum, fibrine, or pus, no thickening, but the different free surfaces of the peritoneum are attached to each other by adhesions. These adhesions are in the shape of threads or of membranes, often of the most extreme tenuity. They are formed of a fibrillated basement substance, the fibrils crossing each other in all directions. Imbedded in the basement substance are cells, some fusiform and stellate, but most of them represented by large nuclei apparently imbedded in the larger fibres of the basement substance. They give the impression of being large branching cells of which the cell-bodies have become fused into basement substance, while the nuclei remains. Plate XXVII. shows part of such an adhesion.

Close to these adhesions the peritoneum may appear normal to the naked eye, but, if it is put in water, very fine threads and membranes will float upward from its free surface. Minute examination shows that the connective-tissue cells are increased in size and number, that the endothelial cells are replaced by cells of a great variety of shapes, and that the thin little threads and membranes on the surface are formed of large branching cells. Plate XXVIII. represents the surface of the peritoneum in such a condition. The large branching cells, which are drawn as if laid flat, really float out from the surface, only adhering by one or more of their ends.

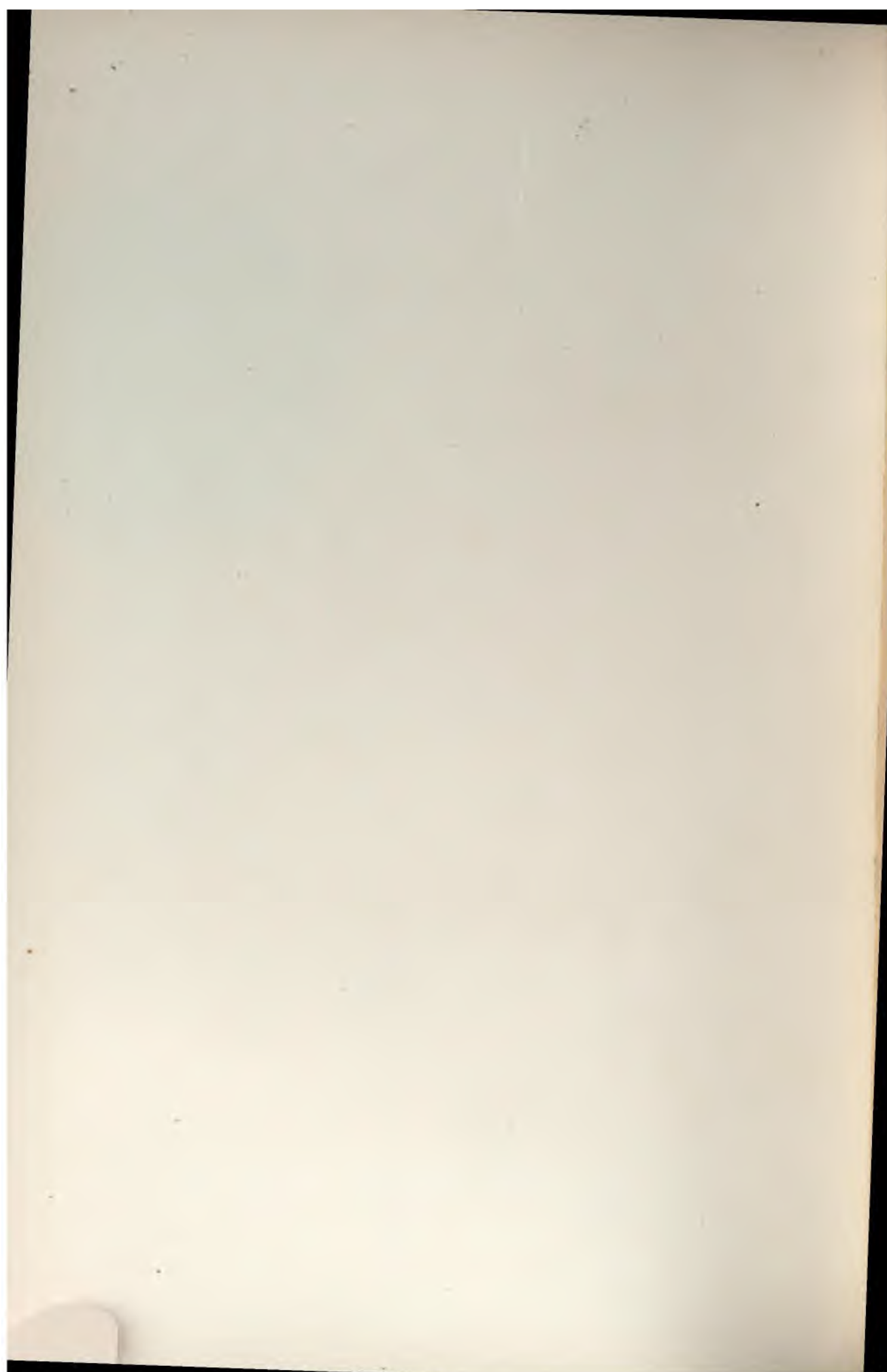
Such a peritonitis with adhesions appears to be a more advanced stage of the forms of cellular peritonitis already described. It is produced in the same way by some constant, but not very energetic irritant. But the inflammation, instead of stopping at the point of a simple new

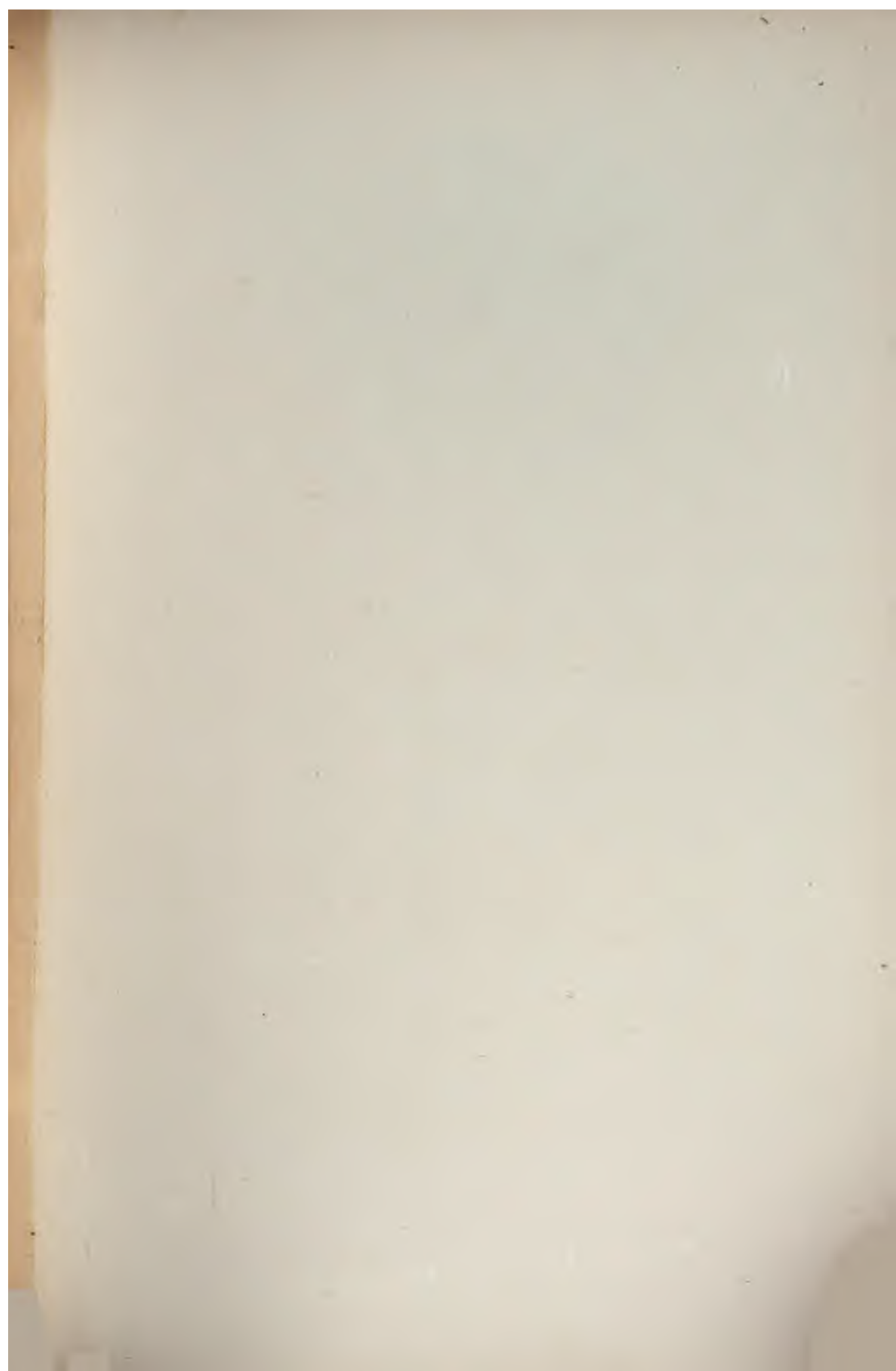


slapell

PLATE XXVII.

*At peritoneal adhesion.
magnified 750 diameters.*





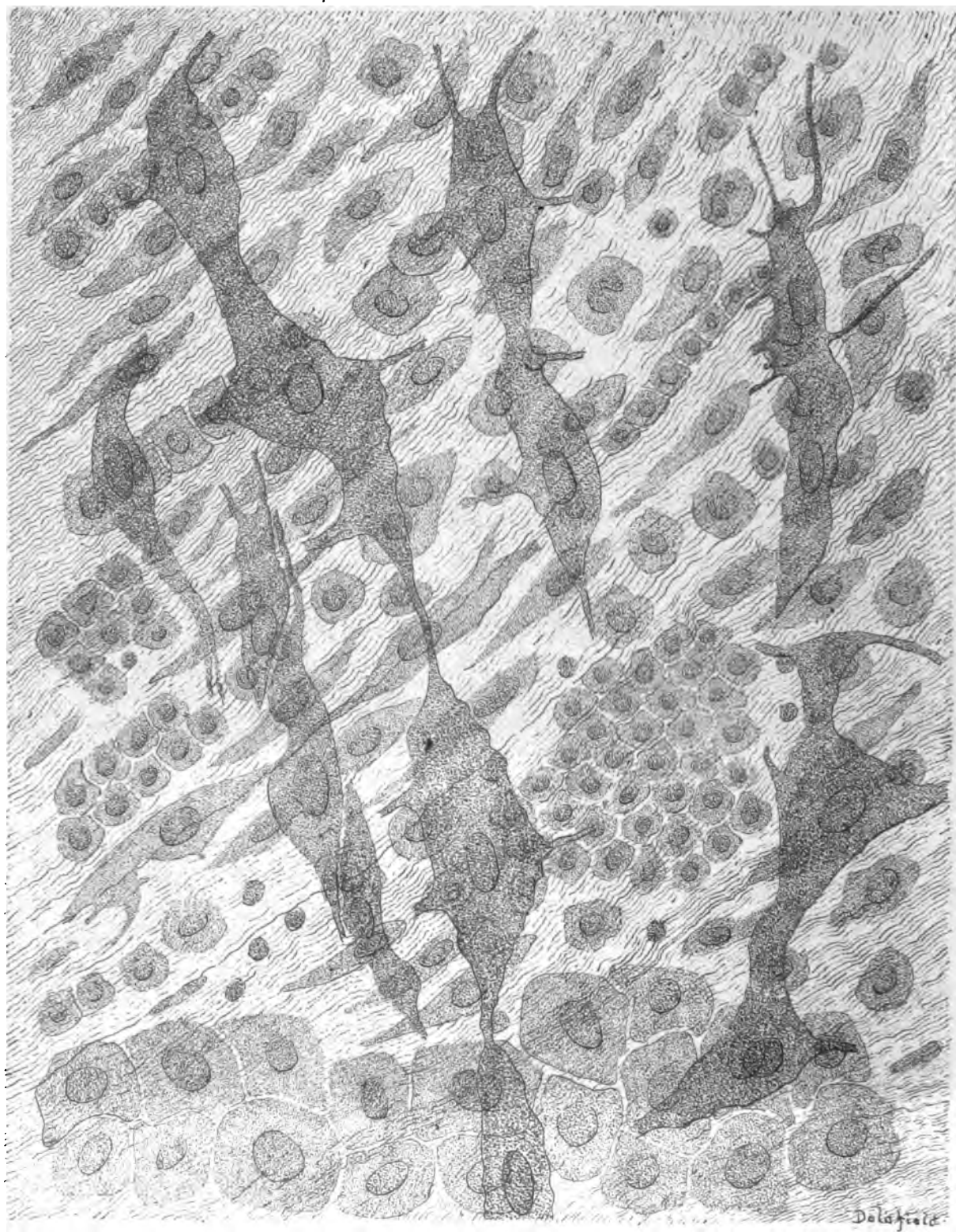


PLATE XXVIII.

*Peritonitis with adhesions. Surface of the peritoneum
magnified 100 diameters.*

growth of cells, continues, and the new cells are changed into membranes.

3. Peritonitis with thickening of the peritoneum. This form of inflammation is not common, and its causation is obscure. The patients are sick and gradually waste away without well-defined symptoms. There is usually vomiting, often diarrhoea. One can feel that there is something wrong about the abdominal cavity, but an exact diagnosis is difficult.

The abdomen usually contains serum, either clear or purulent. The coils of intestine are slightly adherent to each other, or are matted together so as to form an almost solid mass. But the most striking lesion is the thickening of the peritoneum. The parietal peritoneum and that covering the liver, stomach and intestines are all thickened. The surface of the peritoneum is smooth, or is covered with fibrine, or adhesions. The endothelium is always changed. The peritoneum may be as much as an inch thick. It is composed of fibrillated connective tissue, with a moderate number of cells, the cells most abundant near the surface. The change in the peritoneum usually produces changes in the viscera. The liver is small, compressed, and its venous circulation obstructed. The cavity of the stomach is diminished and its mucous membrane is the seat of chronic inflammation. The lumen of the intestines is also diminished in size and their mucous membrane inflamed.

It is necessary to be careful not to confound such cases with diffuse carcinoma of the peritoneum, or tubercular peritonitis. It is sometimes very difficult to distinguish between these three anatomical conditions.

4. Peritonitis with fibrine, serum, and pus. This variety of peritonitis may follow acute peritonitis, may be due to lesions of the abdominal viscera, or may occur without known cause. The following case will serve as an example of the obscure history that belongs to many of these patients. A woman, 36 years old, married, was attacked nine months before her death by a fever which kept her in bed four weeks. At the end of the four weeks she was able to get up and walk about, but never felt really well. For seven months before her death there was pain in the abdomen and gradual increase in its size. There was also a cough with mucous expectoration. I first saw her two months before her death.

She was then feeble and emaciated. Her abdomen was large, but not tender. It did not seem to be distended by fluid, but gave the impression of there being some large, diffuse growth filling up the deeper parts of the abdomen. Her bowels were moved regularly. She remained in this condition gradually losing strength from day to day. Her temperature was sometimes 99 or 101°, but on many days was normal during the twenty-four hours. At the autopsy all the viscera were examined, but there were no lesions except of the peritoneum. This membrane was everywhere moderately thickened and its surface covered with fibrine and pus, while the abdominal viscera were firmly matted together by organized adhesions, so as to form a single large mass. Between the adhesions were shut in numerous collections of purulent serum.

In all the cases permanent adhesions, pus, fibrine and serum are the regular lesions; the adhesions either binding up most of the viscera together, or dividing the abdomen into a number of cavities containing pus and serum. The walls of the intestines are usually soft and easily torn.

The adhesions are formed of bundles of connective tissue crossing and interlacing, and of polygonal and branching cells of many shapes. The thickened peritoneum is also covered and infiltrated with similar cells and with pus-cells.

5. Hemorrhagic peritonitis. This form of peritonitis is seen most frequently as a local inflammation. It affects the peritoneum behind and around the uterus in the female, and that covering the recto-vesical excavation in the male. The affected portion of the peritoneum is covered with layers of thin membrane infiltrated with more or less blood. The membranes are formed of numerous blood-vessels with thin walls; of branching cells, often pigmented; and of a scanty basement substance. It resembles very much the chronic hemorrhagic inflammation of the dura mater, and, as in the latter, the extravasated blood may form tumors of large size.

General hemorrhagic peritonitis I have not seen. Friedreich, however, describes two cases occurring in patients with ascites, who had been frequently tapped. He says that both the parietal and visceral perito-

neum were covered with a continuous membrane of a diffuse yellowish brown color, mottled with small and large extravasations of blood. The membrane was thickest over the anterior abdominal wall. It could be separated into a number of layers. These layers were composed of blood-vessels, masses of pigment, branching cells, and fibrillated basement substance. In many places the extravasated blood was coagulated in the shape of round, hard, black nodules. The entire new membrane could be readily stripped off from the peritoneum, and there were no adhesions between the visceral and parietal peritoneum.

TUBERCULAR PERITONITIS.

Tubercular peritonitis occurs as one of the lesions of acute general tuberculosis, with chronic pulmonary phthisis, with tubercular inflammation of the genito-urinary tract, and as a local inflammation. When it occurs as a local inflammation there is often tubercular pleurisy as a complication.

The gross appearance of the lesions varies with the amount of tubercle produced and with the character of the accompanying inflammatory lesions.

When tubercular peritonitis occurs as one of the lesions of general tuberculosis, the tubercle is usually in the form of small nodules, some of them hardly visible to the naked eye. The only inflammatory changes in addition are an increase of the endothelial and connective-tissue cells, with sometimes a little fibrine. The tubercle is in the form of tubercle-granula. Some writers describe the accumulations of cells on the omentum, such as are figured in Plate XXVI., as tubercle. It seems to me, however, that if we compare the omentum in acute cellular peritonitis and in the different examples of chronic cellular peritonitis, it is evident that the changes are in all the cases essentially the same, and when we find the same lesions accompanying the production of tubercle, they are merely accompanying inflammatory lesions. Tubercle, so far as I know, is never produced alone; there is always at the same time, fibrine, or pus, or serum, or a new growth of connective-tissue cells.

Even the smallest tubercle-granula are not composed of a mere accumulation of cells, but of a regular arrangement of reticulated basement substance with cells imbedded in it, as is seen in Plate XXIX.

The practice of calling every accumulation of small cells tubercle leads to a confusion which is quite unnecessary. It is very easy in animals to excite artificial chronic cellular peritonitis, and, as a result of this, little nodules in the omentum formed by the accumulation of endothelial and connective-tissue cells, but such nodules have nothing to do with true tubercle. It is evident that we can make no real advance in our knowledge of tubercle unless we adhere strictly to some one anatomical lesion as being tubercle. Such a distinct lesion we have in the regular arrangements of basement substance and cells which make up tubercle-granula and diffuse tubercle. All the accompanying inflammatory products must be recognized as accessories only.

In local tubercular peritonitis, although there are many varieties, we may conveniently follow Wilks and Moxon in distinguishing three groups of cases. First, tubercular ascites. The peritoneum is thickened, it is studded with accumulations of tubercle from the size of half a pin's head up to large masses as much as an inch in diameter. Some of these nodules are rounded, others are spread out in flat plates. There are but few adhesions, but there is a large amount of turbid serum. The omentum may be much thickened. Such cases are sometimes hard to distinguish from diffuse cancer of the peritoneum, especially if the tubercle has degenerated. There is often tubercular pleurisy at the same time, and sometimes tubercle in the spleen and lymphatic glands, but the other viscera are usually free.

The symptoms of such cases are much like those of ascites with cirrhosis of the liver.

Second, tubercular peritonitis with fibrine. The peritoneum is studded with tubercular nodules imbedded in the midst of a soft, semi-gelatinous matter which covers the whole of the peritoneum and sticks together its different surfaces. The soft matter is principally composed of fibrine infiltrated with serum. These cases are apt to have very obscure clinical histories. There may also be tubercular pneumonia as a complication.

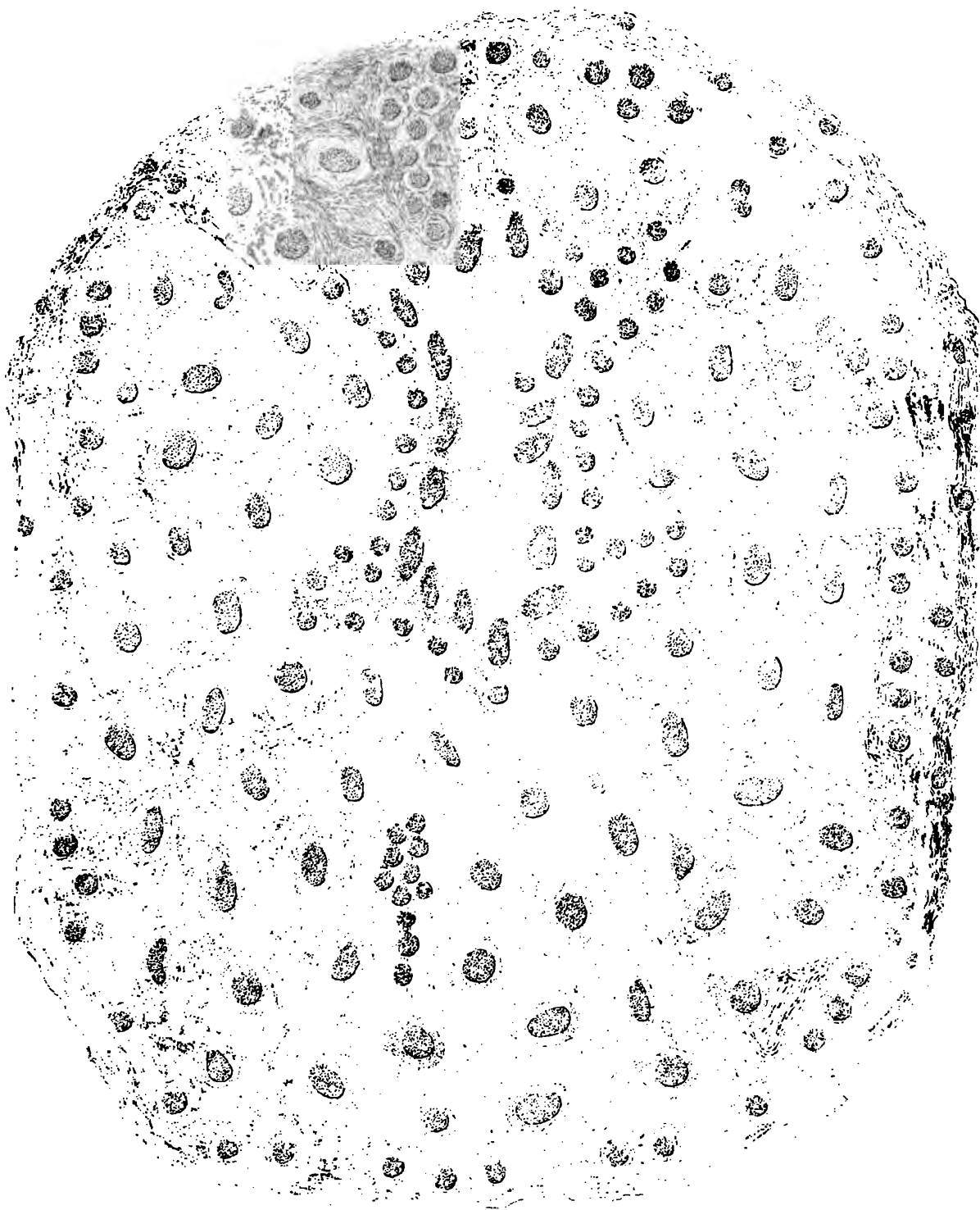
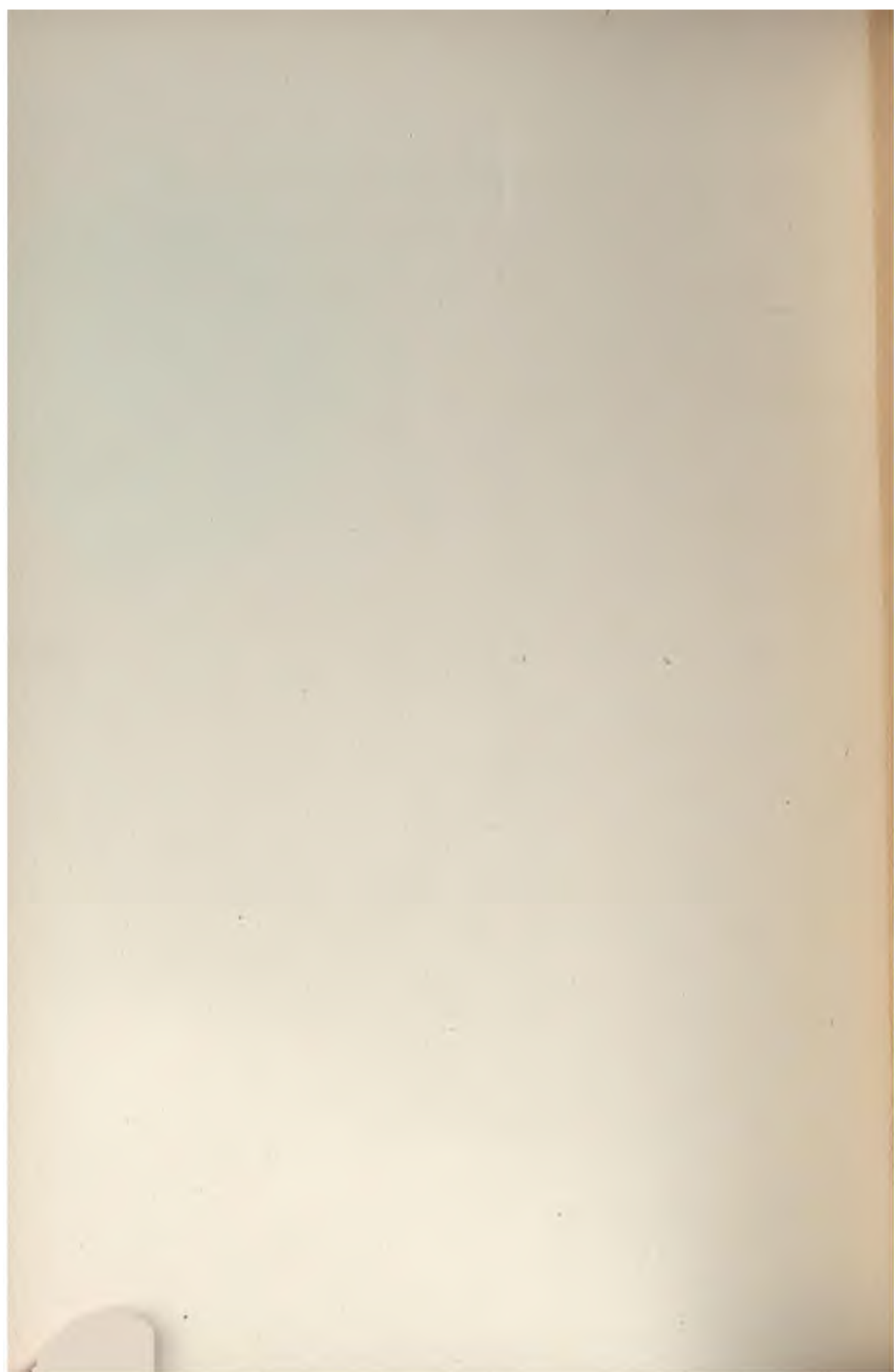


PLATE XXIX.

*Tubercular Bacillus Section of a tubercle granulum.
magnified 1,500 diameters.*



Third, tubercular peritonitis with adhesions. The peritoneum is thickened and there are numerous organized adhesions. All the abdominal viscera are firmly matted together, and there may be collections of pus shut in by the adhesions. The adherent coils of intestine may ulcerate and open into each other. The tubercular nodules are small or large; they are found everywhere in the peritoneum, and also in the adhesions.

Such a tubercular inflammation may be confined to the peritoneum, or there may be also tubercular pleurisy, or pulmonary phthisis, or tubercle in the spleen and liver, or tubercular nephritis, or metritis.

It is evident that the different characters of these different forms of tubercular peritonitis depend not so much upon the different kind of tubercle produced as upon the different kind of accessory inflammation. The presence of cells, or of serum, or of fibrine, or of adhesions, forms the most marked feature in each case.

PNEUMONIA.

THE lungs are composed of bronchi, air-vesicles, blood- and lymph-vessels, and connective tissue; the latter forming part of the walls of the bronchi, the air-vesicles, and the blood-vessels, holding together the different parts of the lung, and constituting the pulmonary pleura.

The anatomy of the lungs has been so often described that it is useless to speak of it here. I must say a word, however, concerning the epithelium of the air-vesicles. In foetal life the air-vesicles are lined with cylindrical cells resembling the epithelium of the bronchi. As the foetus grows older these cells become broader and flatter, until at the time of birth there is a continuous layer of flat, polygonal, nucleated cells lining each of the air-vesicles. As soon as breathing begins, the air-vesicles expand and their walls are stretched to correspond with the increased size of the contained spaces. The epithelial cells which line the air-vesicles must, under these circumstances, either be separated from each other or else the cell-bodies must become broader and flatter, so as to cover more space. The latter seems to be the case. As the child grows up to adult life this thinning of the cell-bodies of the epithelium seems to continue and to be accompanied by an atrophy of some of the nuclei. In adult human lungs, therefore, we find the air-vesicles lined with a very thin homogeneous layer, apparently formed of the flattened cell-bodies, and with nitrate of silver this layer

is shown to be divided up into irregular spaces. In this layer are large nuclei scattered at irregular intervals. There are also, however, in most of the air-vesicles, cells which resemble the fœtal epithelium, with a well-marked cell-body and nucleus. Fig. XXX. shows part of the wall of an air-vesicle covered with its epithelium. The preparation was from a healthy, adult human lung removed from the body one hour after death and treated with osmic acid and hæmatoxyline. It will be seen that there are a number of large nuclei, irregularly arranged, imbedded in a thin layer of finely granular matter. Around some of these nuclei the layer of granular matter may be thicker and more opaque, but there is no well-defined cell-body. There are, however, a few flat cells with a distinct nucleus and cell-body. This is all that can be seen of an epithelium in the adult human lung: a very few flat, nucleated cells, and a considerable number of nuclei imbedded in a thin layer of finely granular matter. If we use nitrate of silver we obtain such a picture as is given in Fig. 154 of Stricker's Manual.

This arrangement of nuclei and cells is spread out over the walls of the air-vesicles, walls composed of a thin layer of connective tissue reinforced with elastic fibres. Imbedded in this connective-tissue membrane is a layer of capillary blood-vessels.

The walls of the air-vesicles are, then, an arrangement of tissues designed for the performance of a special function. It can serve no good purpose to compare them with either mucous or serous membranes. They are simply air-vesicles, and nothing else.

It is customary to class all those inflammatory processes which involve the air-vesicles and the interstitial tissue of the lung under the name of Pneumonia. It is also customary to subdivide pneumonia into the anatomical varieties of catarrhal, croupous, and interstitial pneumonia. To the name of interstitial pneumonia there can be no objection; to the names of catarrhal and croupous pneumonia there are very serious ones. They are the result of comparing the air-vesicles with mucous membranes, structures from which they are entirely distinct. These names seem to me so utterly inappropriate that I prefer to describe the different forms of pneumonia as follows:

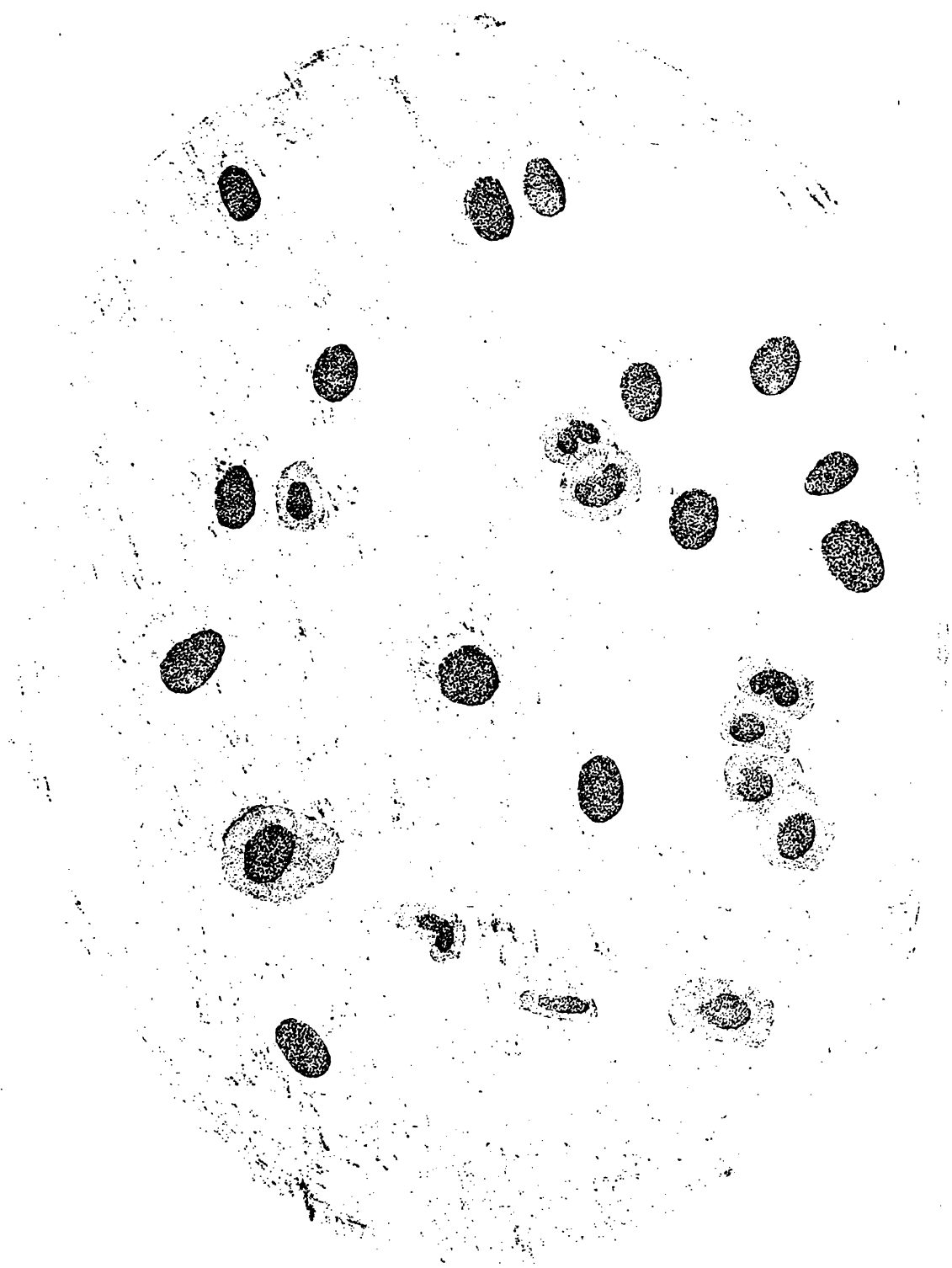


PLATE XXX.

*Epithelium of adult human Lung.
magnified 1300 diameters.*



1. The pneumonia of heart disease.
2. The lobar pneumonia of adults and children.
3. The lobular pneumonia of adults and children.
4. Interstitial pneumonia.
5. The pneumonia produced by pressure on the trachea or bronchi.
6. The lesions of acute tuberculosis, and of acute and chronic pulmonary phthisis.

I. The Pneumonia of Heart Disease.

Lesions of the valves of the heart and dilatation of its ventricles are regularly followed by chronic congestion of the brain, lungs, liver, spleen, alimentary canal, kidneys and subcutaneous connective tissue. In some cases all these viscera suffer; more frequently one viscus will be affected more than the others. Such a chronic congestion is as a rule followed by certain regular changes in the tissues of the affected viscera, so that not only chronic congestion, but also chronic inflammation exist. In old cases the changes due to the chronic inflammation may eventually become more marked than the congestion.

In the lungs such chronic congestion is very frequently followed by marked structural lesions. These lesions have never attracted the attention of clinical observers which their importance demands. One reason, perhaps, for this is that it is very difficult to make out this condition during the patient's life. There are indeed usually cough and an expectoration of mucus and blood, but these may only indicate bronchitis. Dyspnoea exists, but we expect to find it with organic heart disease under any circumstances. The physical signs also are not characteristic. There may be dulness on percussion, but not always, and the hydrothorax, which so often exists, may mask it. If there are any râles, they belong to an accompanying bronchitis. Bronchial breathing and bronchophony are not usually heard, even when the lung is fairly solidified.

And yet the lesion may have the principal share in causing the patient's death. Indeed, if these changes in the lung are once fairly established, the prognosis of the case is hopeless, for the lung can never return to a natural condition, but becomes constantly more unfit for its

work. The pathological recognition of this condition dates back to its description by Virchow in 1847.

“There is a process,” he writes, “which, in spite of its frequency and characteristic appearance, has attracted but little attention. It occurs in patients who have suffered for a long time from asthma or chronic bronchitis, dependent upon disease of the left heart and accompanied by hæmoptysis. Insufficiency and still more frequently stenosis of the mitral valve most frequently produce it. When the thorax is opened, the lungs appear prominent, do not collapse, are compact, heavy, inelastic, crepitate but little, and have frequently a peculiar yellow, brown, or reddish-brown color. On section the tissue is denser, and at many points are red spots, dark colored at their centres, lighter at their edges, also other spots of a brown, rusty, or black color. The parenchyma between these spots is seldom of its normal color, but is yellow, orange, or rust-colored. A yellow or brown, slightly frothy fluid, in many cases, escapes on section abundantly. Microscopical examination of such lungs shows only the normal tissue, with extravasated blood in different stages of transformation into pigment. In the neighborhood of the points of extravasation the epithelial cells of the alveoli are stained yellow, and it is easy to see that these cells are identical and continuous with the colorless pavement epithelium. There are also other cells retained in the same way, but containing in addition granules of pigment. These granules show all gradations of color from yellow to black. Finally the cells may be destroyed and the pigment left free. In addition, extravasations of blood take place in the interstices of the tissue, and these become changed into pigment.

“This lesion of the lungs is produced by a chronic hyperæmia. There is at the same time chronic bronchitis with hyperæmia and thickening of the mucous membrane of the bronchi, and dilatation of their calibre. Any thickening of the walls of the air-cells is hypothetical. I consider the most appropriate name for this condition of the lungs to be brown or pigment induration.”

These names of brown or pigment induration have since been generally adopted.

The attention of Virchow seems to have been directed especially to the hyperæmia and the formation of new pigment.

Of late years several observers have called attention to the dilatation of the capillaries of the air-vesicles, and to the hypertrophy of the walls of the air-vesicles. The thickening of their walls is due to a new growth, partly of connective tissue and partly of smooth muscle.

A single case is described by Orth in which there was also pigment in the capillaries and some of the smaller vessels.

The lesion is a common enough one in New York, occurring in a large number of the cases of organic heart disease, especially with mitral stenosis.

The lungs are usually small, not large and prominent as in Virchow's cases. There are often old pleuritic adhesions connecting the pulmonary and costal pleura and binding together the different lobes of the lungs. In the pleural cavities is a moderate amount of clear serum. When the lungs are removed from the thoracic cavity their small size is still more apparent. The pulmonary pleura is thick and opaque, and gives the surface of the lung a grayish color, mottled by the pigment in the lung-substance beneath it. When the lungs are cut open, the color and texture are very characteristic. The texture is dense and leathery; it does not have the elastic feeling of a normal lung, nor does it contain as much air. On section very little blood or serum escape, and the cut surface is almost dry. The color of the lung is peculiar—a sort of yellow-pink, quite different from the color of a normal lung. This pink is mottled with spots of brown or black pigment, varying in amount in different cases. These changes embrace all the lobes of both lungs.

The leathery consistence of these lungs is often somewhat changed by an hepatization of parts of the lung. This hepatization may involve portions of the different lobes or an entire lobe. The solidification may be partial or complete, and the texture of the lung will be accordingly less or more firm. It is a perfectly smooth hepatization, not resembling that of acute lobar pneumonia. I have never seen it in any stage except that of red hepatization.

There may be hemorrhagic infarctions scattered through the lungs,

and sometimes these are surrounded by a zone of pneumonia; but this pneumonia is different from the red hepatization of which I have just spoken.

The gross appearance of the lungs may be modified by the coexistence of emphysema or œdema.

If the lungs are emphysematous they will be large, instead of small, and they will contain more air and be of a less leathery texture, although their general appearance will be as described above.

If the lungs are œdematous, they will be infiltrated with serum, and consequently large and heavy.

Microscopical examination of lungs which exhibit the gross appearances mentioned shows that there are four separate pathological conditions existing:

(1.) Hypertrophy of the capillaries. This change exists in very different degrees in different cases. In some lungs the capillaries are both dilated and increased in length, so that they appear tortuous and project in loops into the air-vesicles. If they are injected artificially they occupy so much space as to obscure all the other tissues. In other cases the capillaries can hardly be said to be dilated at all; even when injected artificially, they are not large. Between these two extremes of great dilatation and no dilatation at all we find every gradation. In all cases, however, if we inject these lungs artificially, the injection requires more pressure than in a normal lung and the vessels are not as uniformly filled. Although, therefore, the mechanical cause—the obstruction to the passage of blood into the left auricle—would seem likely simply to cause an accumulation of blood in the pulmonary veins and their capillaries, and then a dilation of these vessels, yet the mechanism does not seem to be quite so simple as this. There does not seem to be necessarily any real accumulation of blood in the lungs, but rather a retardation of the circulation. The lungs do not at any one time contain more blood than do normal lungs—if we can judge from post-mortem appearances, they may contain less—but the blood passes through them more slowly than it should. If this view be correct, then the large size of the capillaries depends, not upon their dilatation by the pressure of the blood, but upon an hypertrophy of their walls.





PLATE XXXI.

*The Pneumonia of Heart Disease.
magnified 300 diameters.*

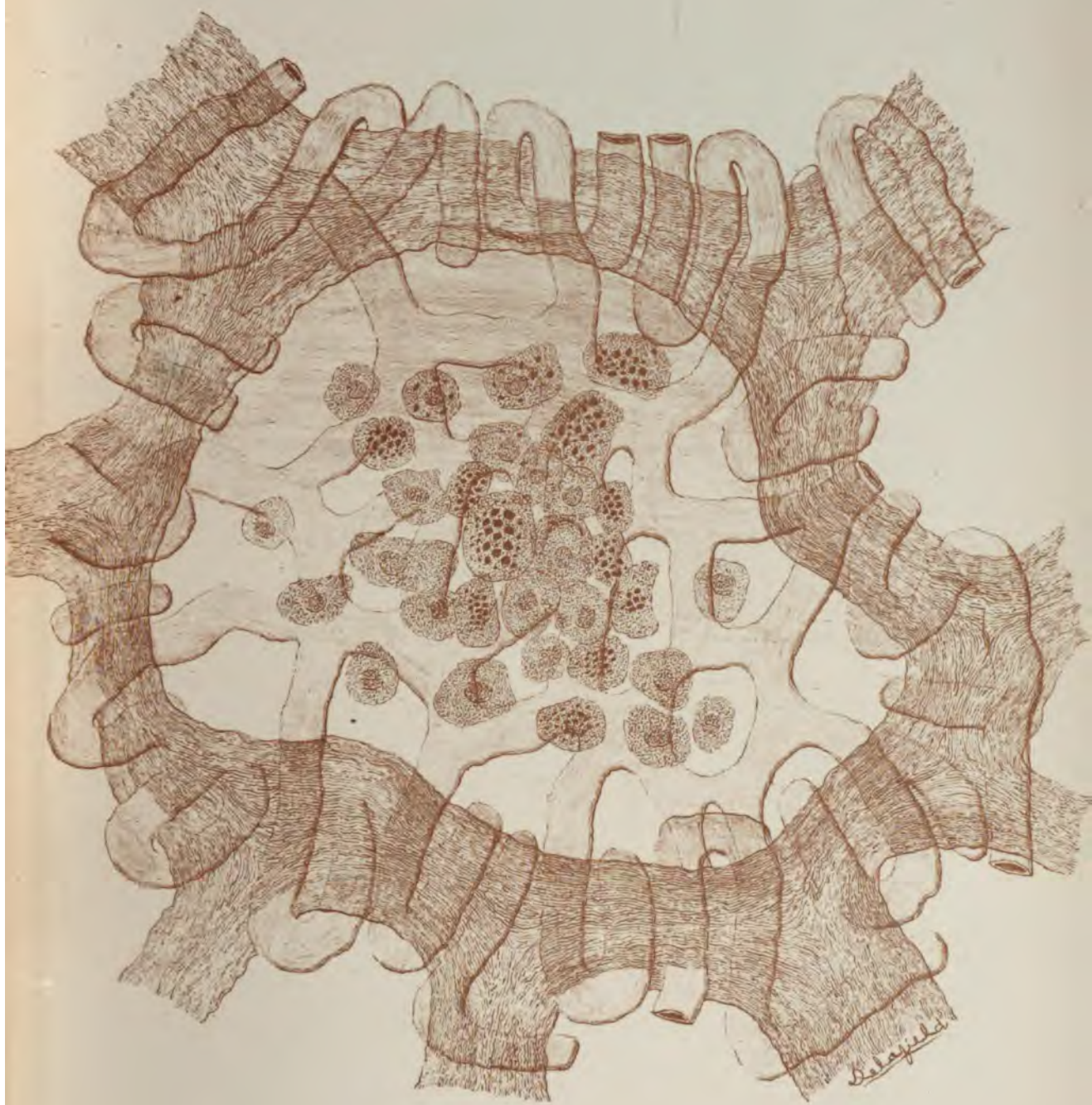


PLATE XXXII.

*The Pneumonia of Heart Disease:
magnified 750 diameters.*



The increased size of the capillaries, as well as the other lesions yet to be described, are due to a change in the nutrition of the lungs produced by the slow circulation of the blood through them, and these changes may fairly be classed with other chronic inflammations.

(2.) Hypertrophy of the walls of the air-vesicles. This lesion also varies in extent. The walls of the air-vesicles may not be thicker than those of a normal lung, or they may be moderately thickened, or very much thickened. When hypertrophy of the walls of the air-vesicles does exist, it is due partly to the increased size of the capillaries, partly to a new growth of smooth muscle, and partly to a new growth of connective tissue.

(3.) Formation of pigment. The pigment is usually of black or brown color, in the form of granules and small masses, or of a diffuse staining. It is deposited in the walls of the air-vesicles, in the interstitial tissue, and in the cells found within the air-vesicles. Orth describes a case in which there was pigment within the capillaries. It seems probable that this pigment is derived from the blood circulating slowly in the capillaries and from that extravasated in the air-vesicles and the interstitial tissue. It is very likely that some of the red blood-globules are taken inside of other cells, and their pigment then transformed, and that others give up their coloring matter more directly. The whole amount of pigment varies in different cases.

(4.) Filling up of the air-vesicles with polygonal nucleated cells and red blood-globules.

It has been already stated that in the normal lung the epithelium is represented by a continuous thin layer of granular matter in which nuclei are imbedded at irregular intervals, and by a small number of polygonal nucleated cells. In pigment induration of the lung these polygonal nucleated cells increase enormously in number, and take the place of the nucleated membrane. We find, then, the entire walls of air-vesicles covered with a continuous layer of flat nucleated cells, either transparent or stained yellow, or containing granules of pigment. There are also similar cells free in the cavities of the air-vesicles and even in sufficient numbers to fill them. These free cells, however, are larger, often more rounded and coarsely granular, and frequently con

tain more pigment than do the more regular cells which line the air-vesicles.

Plate XXXI. is from a case in which the thickening of the walls of the air-vesicles and the production of new epithelium were the most marked lesions. The capillaries were not dilated, nor was there much new pigment. The drawing is made out of proportion, the air-vesicles being magnified 300 diameters and the epithelium 700 diameters.

Plate XXXII. is from a case in which the increase in the size of the capillaries was marked. It represents a single air-vesicle, not injected, magnified 750 diameters.

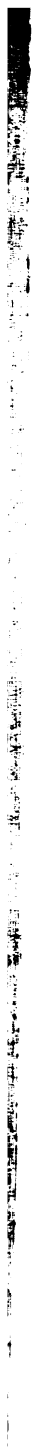




PLATE XXXIII.

*Acute Lobar Pneumonia, Red Hepatization,
magnified 750 diameters.*

II. The Lobar Pneumonia of Adults and Children.

THIS disease prevails regularly in New York and furnishes from about three to four per cent. of the yearly mortality. Since 1873 the mortality has not been less than four per cent. This is the number after deducting the deaths from pneumonia in children under five years, in most of whom the disease is not lobar but lobular. What proportion of children under five years old have lobar pneumonia, I am unable to ascertain.

The larger number of the deaths occur in the three spring months—March, April and May—and the three winter months—December, January, and February—corresponding to the period of cold and wet weather. Still in the hottest months—June, July, and August—there are from fifteen to forty deaths a month.

Persons between the ages of ten and twenty furnish the smallest, and those between forty and fifty the largest number of deaths.

Lobar pneumonia affects large portions of the lung tissue, either the whole of one lobe, or of one lung, or portions of both lungs. It often begins in one lobe and then extends to the rest of the same lung. In rare cases nearly the whole of both lungs is involved at the very outset of the disease.

It is customary to divide the lesions into the stages of Congestion, Red Hepatization, Gray Hepatization, and Resolution.

(1.) Congestion. Of this stage we have but little knowledge derived from actual observation. It seems probable, however, that there is such a stage characterized by an increase of the blood circulating in the vessels.

(2.) Red Hepatization. The lung loses its natural consistence and becomes solid. Firm pressure causes it to break down. Its color is red, sometimes mottled with hemorrhagic spots of a deeper hue. A section looks as if the lung was composed of a multitude of small granules closely packed together, each granule corresponding to an air-vesicle. If the lung is seen soon after death, the section is dry; in a short time, however, part of the inflammatory products liquefies and the section is

covered with a thick, grumous, reddish fluid. The larger bronchi are congested and coated with mucus, or dry, or filled with firm, yellow, fibrinous cords. The pulmonary pleura is coated with fibrine.

In the earlier stages of red hepatization, before the air-vesicles are fairly filled, we find in them fibrine, epithelial cells, pus-cells and red blood-globules. The vessels of the lungs are full of blood, the walls of the air-vesicles are unchanged. Although the blood-vessels are full of blood, it is not coagulated, and the vessels are easily filled with an artificial injection after death. The fibrine is present in variable amount, sometimes forming a large part of the inflammatory products, sometimes hardly visible. It has seemed to me that large amounts of fibrine are usually associated with severe constitutional symptoms. It usually coagulates in the form of a net-work. The epithelial cells also are found in variable proportion. They are more numerous in children, in persons already suffering from emphysema and bronchitis, and in those who have chronic phthisis. They are often swollen and coarsely granular. The pus-cells are always numerous, often obscuring both fibrine and epithelium. The red globules may only be found in small numbers, or they may completely fill some of the air-vesicles. When the stage of red hepatization is complete, the air-vesicles are completely filled with these products of inflammation. Plate XXXIII. represents a single air-vesicle, only partly filled with the products of inflammation. The specimen was from a person who died forty-eight hours after the initial chill. The blood-vessels are injected artificially.

The smallest bronchi are filled with the same products of inflammation as are the air-vesicles—fibrine, pus, and epithelium. The same products may fill the medium-sized and even the largest bronchi. Plate XXXIV. represents a vertical section of the wall of a smaller bronchus; there is a layer of fibrine and pus coating its inner surface and a similar layer lifts up and separates the epithelium from the stroma beneath.

I have seen one case of lobar red hepatization characterized by the presence of large masses of bacteria within the air-vesicles. The patient was a woman thirty-two years old, a laundress by occupation. She was perfectly well until five months before her death. At that time she began to have a slight cough with expectoration, which con-

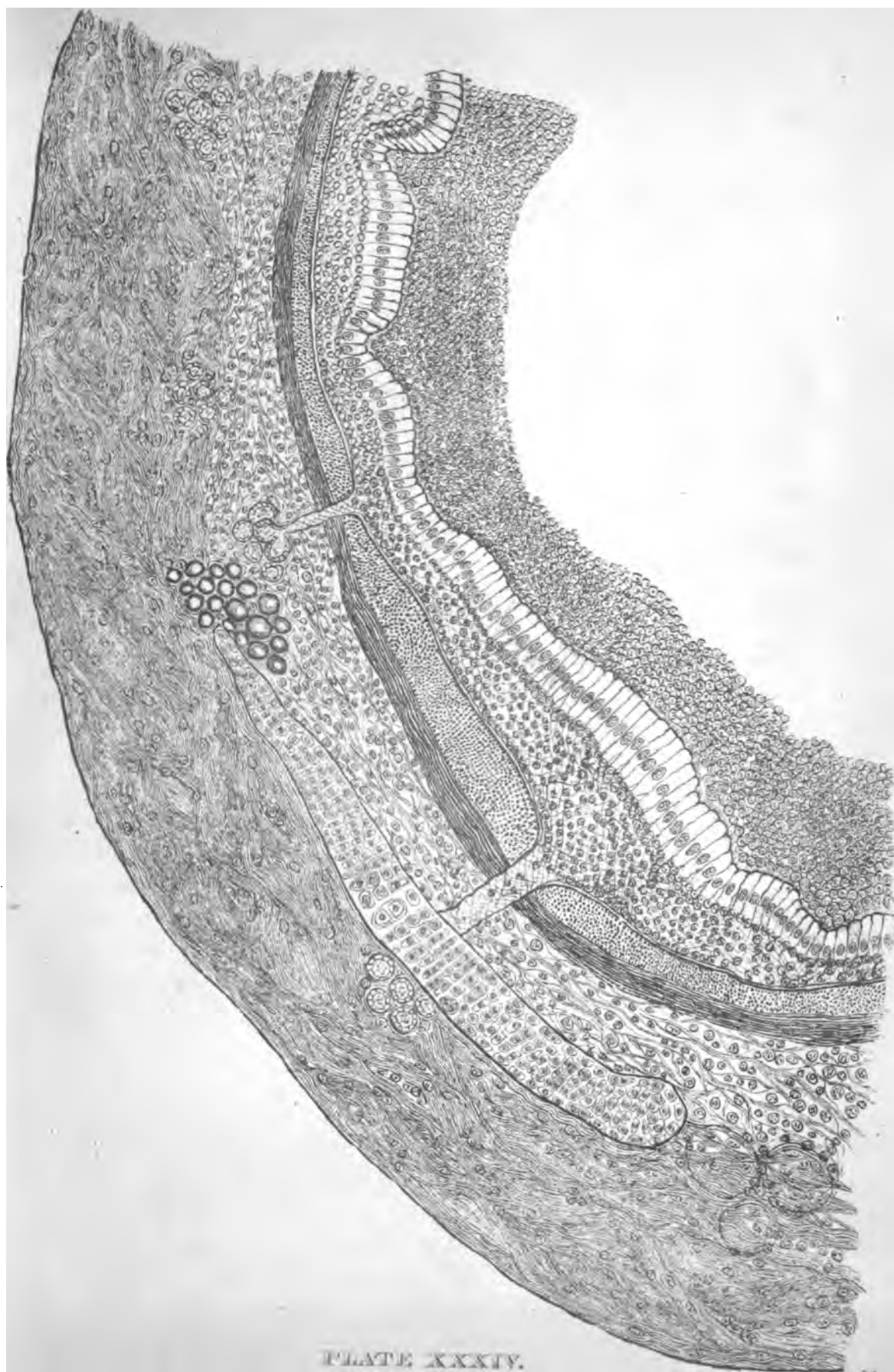
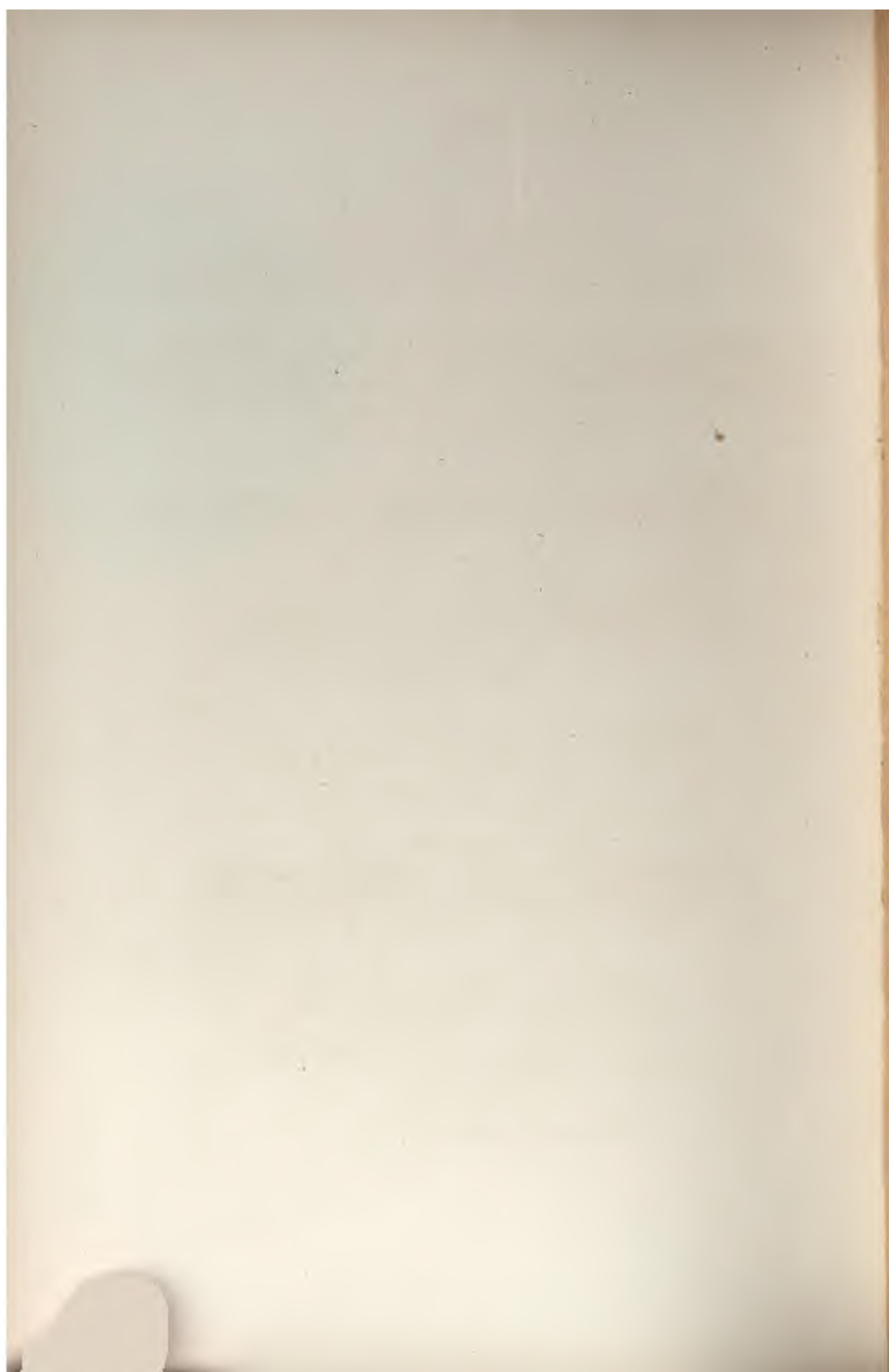


PLATE XXXIV.

Compos. Bionchitis with Delai. Succum.
magnified 500 diameters.

W. H. H. 1892





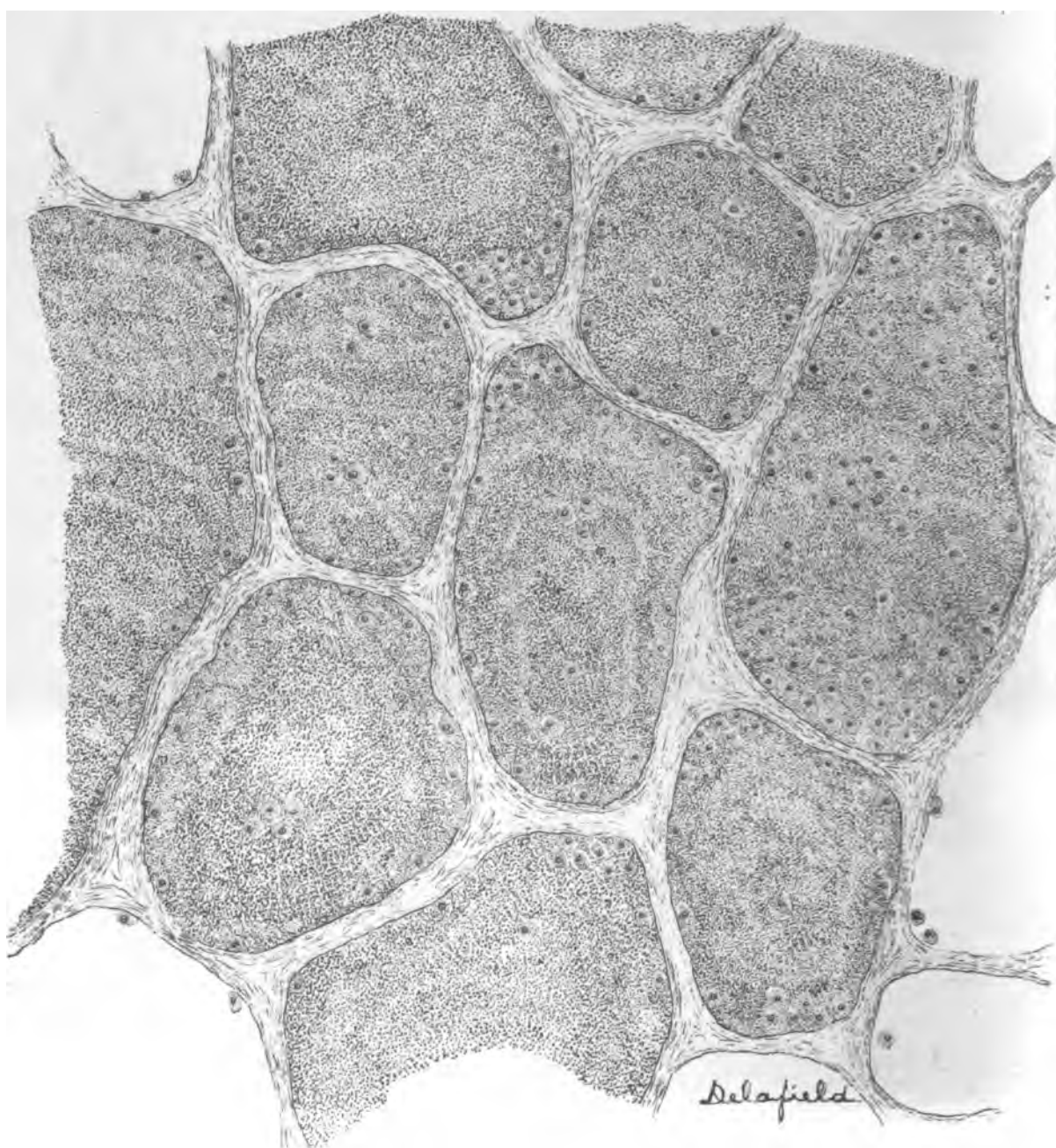


PLATE XXXV.

Bostrychia Americanana
magnified 300 diameters.

tinued until her present attack. Eleven days before her death, after being exposed to cold and wet, she had several rigors followed by high fever. The next day she began to have pain in the left side of the chest. On the fifth day she began to vomit, and after that time vomited everything she ate. I saw her first on the eighth day of her disease. Her temperature was then 106° , pulse 122, respiration 42; there was a good deal of pain and dyspnoea; the woman looked very sick. Over the left upper lobe there was dulness, bronchial breathing, and coarse and subcrepitant râles; over the rest of both lungs coarse râles. She became rapidly worse, with high temperature, delirium, brown and dry tongue, and died on the eleventh day of the disease. At the autopsy there were no lesions except of the lungs. The bronchi of both lungs were congested and coated with muco-pus. The left upper lobe was in the condition of red hepatization, the pleura coated with fibrine. The lung did not look exactly like most specimens of red hepatization, being moister and less firm. The air-vesicles were found to be filled with large masses of micrococci, not affected by acetic acid or chloroform. Mixed with these was a moderate amount of pus, epithelium, and fibrine. See Plate XXXV.

The duration of the stage of red hepatization seems to be quite variable. About one-fourth of the autopsies of pneumonia which I have made have shown red hepatization existing at the time of death. In these cases the duration of the disease has varied from twenty-four hours to eleven days, dating from the initial chill.

(3.) Gray Hepatization. The stage of red hepatization passes into the gray gradually, the lung being first mottled red and gray and then completely gray. Except for the difference in color, a difference which seems to depend partly on the diminished congestion, and partly on the loss of color of the red globules already extravasated, the condition of the lung remains almost the same. The products of inflammation—the pus, fibrine, and epithelium—have much the same appearance. Some granular degeneration of them, however, is usually apparent. The epithelium and pus-cells become swollen and filled with coarse granules, and there may be coarse granules in the fibrine. The air-vesicles are more completely filled and packed with the inflammatory

products. Plate XXXVI. shows one air-vesicle and parts of others from a lung in the condition of gray hepatization. The lungs have been in the mottled condition of partly red and partly gray hepatization in nearly half of my autopsies of pneumonia, so that in New York at least the end of the period of red hepatization is a favorite time for death. The time of the disease, however, at which the lung is found mottled red and gray is very variable. I have found it from the second to the eighteenth day of the disease. The lung was completely gray at the time of death in only about one-fourth of my cases. It was found in this condition from the fourth to the twenty-fifth day of the disease.

(4.) Resolution. I have very little actual knowledge of this stage. It is evident that in persons who recover from pneumonia, the solid products which fill the air-vesicles must in some way become liquefied so that they can be absorbed or coughed out. There are also a moderate number of autopsies in which the lungs look like gray hepatization, but are softer, and covered with grumous fluid when cut. Of course this condition must not be mistaken for the post-mortem softening which may affect any gray hepatization.

In such soft lungs the air-vesicles still contain the products of inflammation, but are not so closely packed with them. There is not nearly as much fibrine, but a great deal of coarse granular matter; the pus-cells and epithelial cells are large and filled with coarse granules, or broken and fragmentary. One receives the impression that all the inflammatory products are degenerating and breaking down; and it seems probable that this is the way in which resolution is effected during the life of the patient.

There are some exceptions to this ordinary course of lobar pneumonia.

(1.) The lung after reaching the stage of gray hepatization may remain in that condition for weeks, without any attempt at resolution. When this is the case, the lung presents a peculiar appearance after death. It is very firm and dry; its color is rather white than gray. The air-vesicles are found completely filled with fibrine, pus, and epithelium. These retain their shape and can be recognized in some air-vesi-

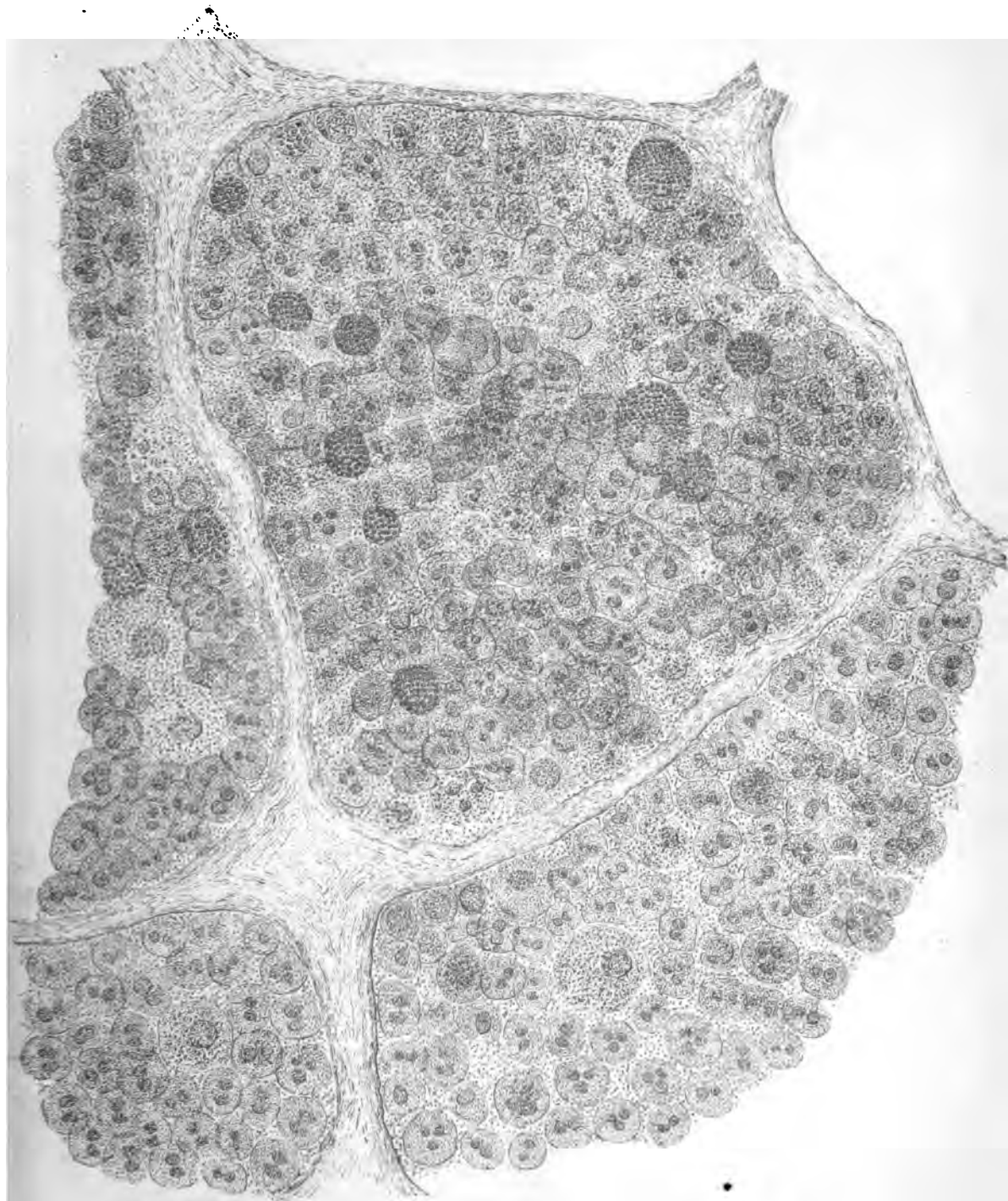
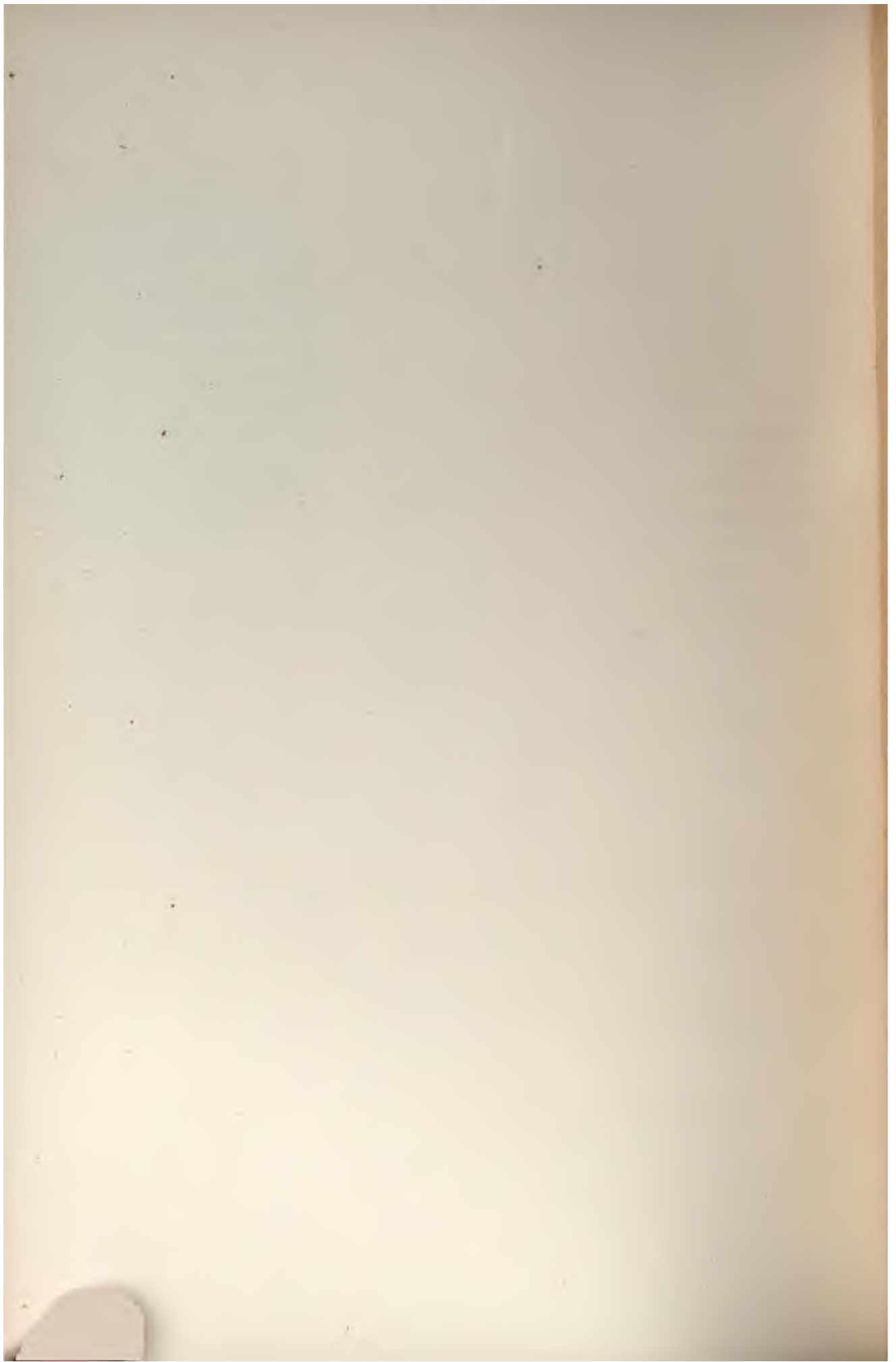


PLATE XXXVI.

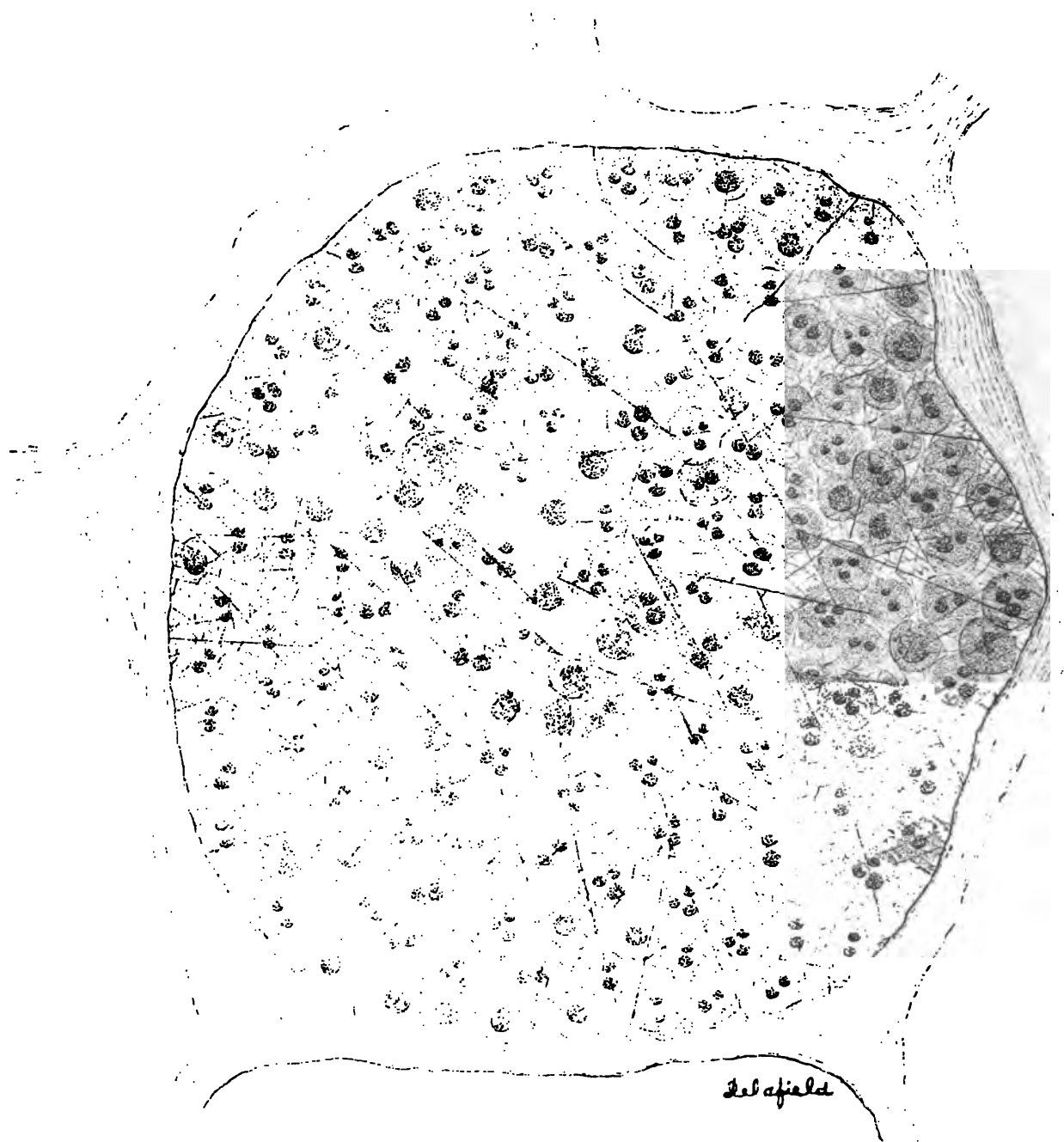
*Teste. Lobes Pneumonia, Grey Hepatisation;
magnified 750 diameters.*



cles, while in others they are changed into a mass of granules. This is the nearest approach to cheesy hepatization that I have seen in lobar pneumonia.

(2.) Portions of the hepatized lung may die and become separated from the surrounding lung tissue, so as to form irregular cavities containing portions of necrotic lung tissue. In the earlier stages of this change, which I have seen, I have been struck by the large amount of fibrine present. All through the hepatized lung the air-vesicles are filled with coagulated fibrine, while pus and epithelium are scanty. There seems to be a necrosis of portions of the hepatized lung, either from complete stasis of the blood, or from the presence of the large amount of fibrine on the vessels. In the later stages the cavities contain pus in addition to the necrotic lung tissue and look more like ordinary abscesses.

(3.) Portions of the hepatized lung may become not only necrotic, but gangrenous, and such portions may be either small or large. Their causation is very obscure.



III. The Pneumonia which occurs after Surgical Operations and Injuries.

The etiology of this variety of pneumonia is very obscure. The disease is developed either a few days after the operation or injury, or not until the patient has been confined to bed for some time. The existence of the inflammation may be indicated by well-marked constitutional symptoms, or such symptoms may be entirely absent. There may be severe rigors followed by a well-marked febrile movement, rapid breathing, dulness on percussion, crepitant râles, and bronchial breathing; or all these symptoms will be wanting, and the lesion is only discovered at the autopsy.

The amount of lung inflamed may be small or large; but the central portions of the lung are those usually involved. The rest of the lung is apt to be congested and œdematous. No matter how small the hepatized portions of lung, they have the characters of a diffuse inflammation. They do not look as if the inflammation began in the bronchi or in the blood-vessels. The suspicion arises, of course, that such a pneumonia may be produced by irritating matters inhaled through the bronchi, or lodged in the vessels as emboli. But the lungs do not look so; there is no appearance of foci; the hepatization has the same diffuse character, the same color and general appearance that it has in idiopathic lobar pneumonia. There is, however, very often catarrhal inflammation of the larger bronchi with the pneumonia.

When we examine the hepatized lung, we find the air-vesicles are filled principally with fibrine and pus; with these are a few epithelial cells and red blood-globules. There is no essential difference between the lesions and those found in idiopathic lobar pneumonia. Plate XXXVII. represents a single air-vesicle full of fibrine and pus. The specimen was taken from a woman who died on the fourth day after receiving a severe blow on the head, which produced laceration of the brain.

IV. The Lobular Pneumonia of Children and Adults.

Most of the examples of lobular pneumonia in adults which I have seen have occurred in persons suffering from chronic bronchitis.

Chronic bronchitis, either with or without emphysema, is usually attended with an increase in the number of the epithelial cells of the air-vesicles. Some of these cells are found in position, others are detached and free within the cavities of the vesicles. The production of new cells is not, however, sufficient to fill the air-vesicles or produce red hepatization. But sometimes a patient, already suffering from such a chronic bronchitis, becomes worse. There is an exacerbation of the bronchitis, and the inflammation extends to the lungs. We then find a number of hepatized lobules scattered through one or both lungs. These lobules are not very sharply outlined; they are usually in the condition of red hepatization, resembling the same condition in lobar pneumonia, but giving a smoother surface on section. Minute examination shows that the air-vesicles contain epithelium, pus, fibrine and red blood-globules. The epithelium is always abundant, many of the cells are well formed, others swollen and granular. The pus-globules are present in considerable numbers, and are usually round and well formed. The fibrine varies in amount in different lungs and in different air-vesicles in the same lung. It is not as abundant as in lobar pneumonia. The red blood-globules also vary in number in different cases. See Plate XXXVIII. The larger bronchi are usually trabeculated, and contain muco-pus; the smaller bronchi contain pus and detached epithelium.

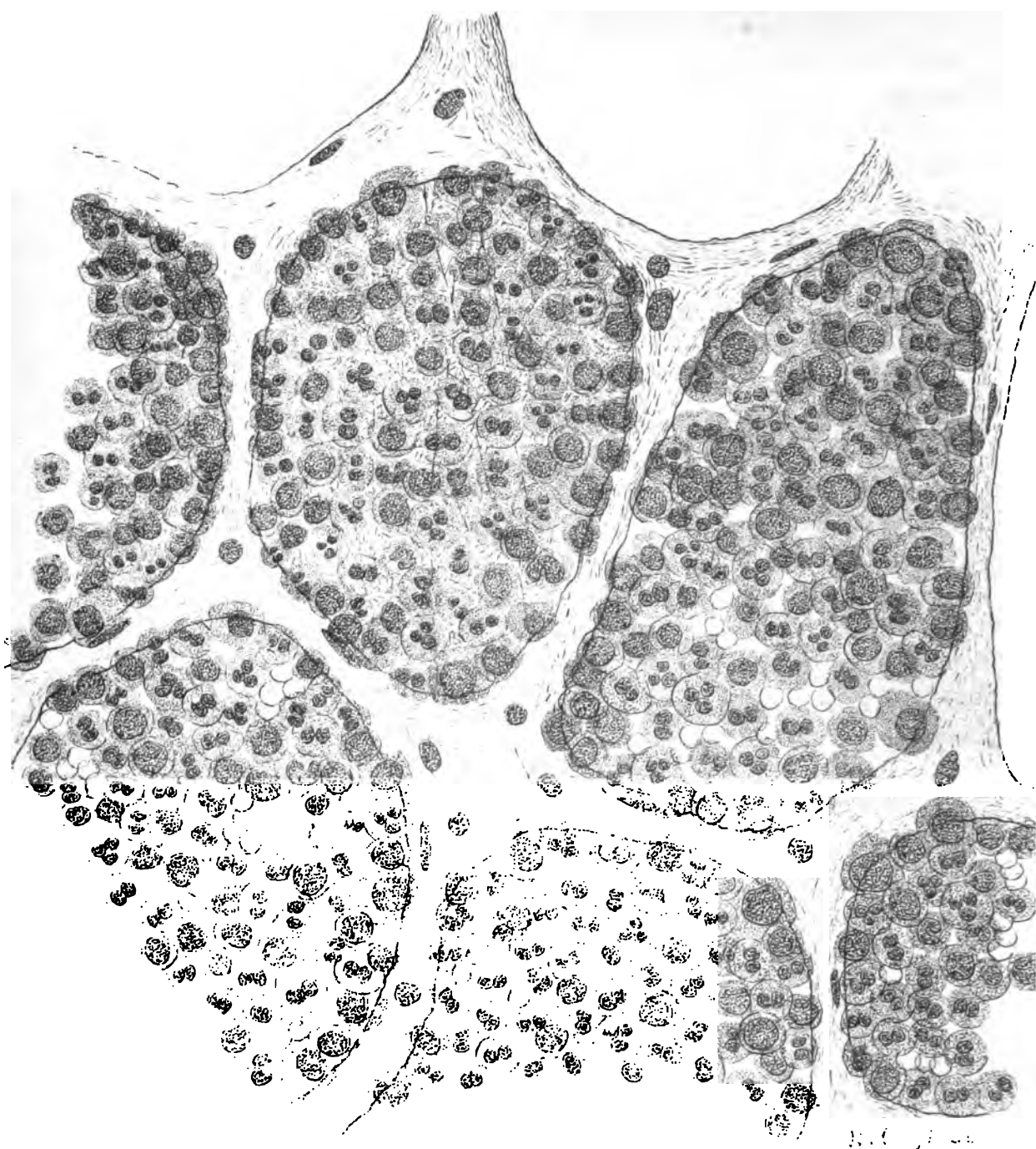
Acute inflammation of the lungs in children is either lobar or lobular. The lobar pneumonia of children resembles in all respects that of adults, both in its gross and minute anatomy. There are, usually, however, a larger number of epithelial cells, and the interstitial connective tissue is more infiltrated with pus and fibrine.

The lobular pneumonia of children, however, is something quite different. It is essentially a broncho-pneumonia, not merely in the sense that the bronchi are inflamed as well as the lung tissue, but that the pneumonia is dependent on the bronchitis. In lobar pneumonia it seems as if a large part of the lung, including both air-vesicles and bronchi, become inflamed at the same time. But in lobular pneumonia the inflammation appears to begin in the bronchi and then extends to the groups of air-vesicles belonging to the inflamed bronchi, and the



PLATE XXXVIII.

L. totus *Polymorphus* of *Abert*.
monogynus *dit* *chambers*



bronchitis continues to be an important factor in the inflammatory process.

A large number of the cases of lobular pneumonia occur in the course of whooping-cough, measles, and scarlatina, diseases which are regularly complicated by bronchitis. Other cases occur with bronchitis due to exposure to cold, and in children who are badly nourished from any cause.

The course of the disease, also, is very different from that of lobar pneumonia. It does not run a regular course, terminating in a few days in death or resolution, but may be protracted for weeks or months before the patient dies, or before the products of inflammation are absorbed. Cheesy degeneration of the hepatized portions of lung and interstitial pneumonia are also much more common with lobular than with lobar pneumonia.

If we look at the lungs of a child in whom lobular pneumonia has existed for only a few days, we find the pulmonary pleura either normal, or coated here and there with a little fibrine.

The large bronchi are congested and coated with mucus; the smaller bronchi are congested, their walls are often thickened, and they may contain pus.

Scattered through one or both lungs are hepatized globules, which may be large or small, few or numerous, scattered or crowded close together. These hepatized lobules may be of a light or deep red color, or pinkish-gray, or of a livid, bluish color. They may be so solid as to stand out sharply defined from the surrounding lung tissue, or only partly hepatized and blending gradually with the adjacent lung. On section they are not granular like lobar pneumonia, but smooth. Sometimes the hepatized lobules have their red color mottled with white spots or lines marking the position of the bronchi.

Such hepatization is not to be confounded with the condition of atelectasis, when groups of air-vesicles are simply collapsed. But sometimes there appears to be an inflammatory process complicating this condition of collapse.

The rest of the lung around the hepatized lobules may appear

normal, or be congested, or be of firmer and denser consistence, or be emphysematous.

Minute examination of the hepatized lobules shows that their condition is not always the same. In some cases the air-vesicles are filled principally with pus; in other cases, almost entirely with epithelial cells; in other cases, with both pus and epithelium; in others, with pus, epithelium, and fibrine. The capillaries in the walls of the air-vesicles are usually large and prominent, and there are red blood-globules in the air-vesicles. The degree in which the air-vesicles are filled also varies; they may be only partly occupied by the inflammatory products, or they may be so closely packed with them that the walls of the vesicles cannot be distinguished. The rest of the lung tissue, although it is not hepatized, shows some change, for the epithelial cells appear larger and more distinct than they do in the healthy lungs of children of the same age. Plate XXXIX. shows part of a hepatized lobule from a case of scarlatina. The air-vesicles contain pus, epithelium, fibrine, and red blood-globules.

Plate XL. shows part of a hepatized lobule from a case of whooping-cough. The air-vesicles are partly filled with epithelium, and the epithelial cells are especially prominent on the edges of the walls of the air-vesicles.

In many cases of lobular pneumonia there are well-marked changes in the walls of the bronchi. Not only are the larger bronchi congested and coated with mucus and the smaller filled with pus and epithelium, but their walls are thickened and infiltrated with cells. Sometimes the thickening is so great that the bronchi appear like white lines and dots to the naked eye when the lung is cut. This thickening of the bronchi seems to be due merely to an infiltration of pus, or, in other cases, to a growth of connective-tissue cells in addition to the pus.

Plate XLI. represents a vertical section of part of the wall of a small bronchus thus thickened. The epithelium lining the bronchus is swollen and deformed, and the wall of the bronchus beneath is thickly infiltrated with cells. The connective tissue between the lobules may be in the same way infiltrated with cells.

Many of the patients die while the lungs are still in the condition of

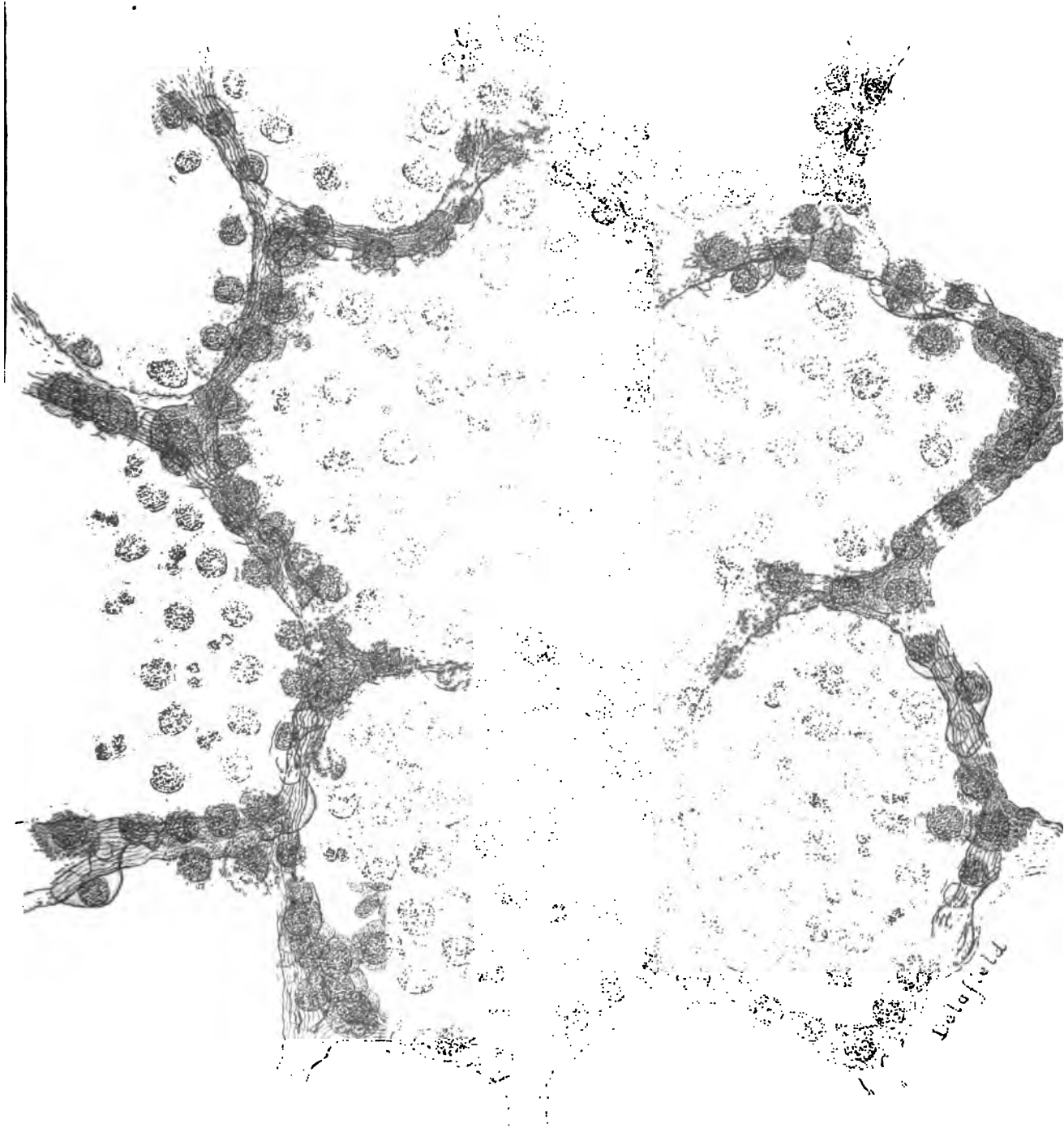
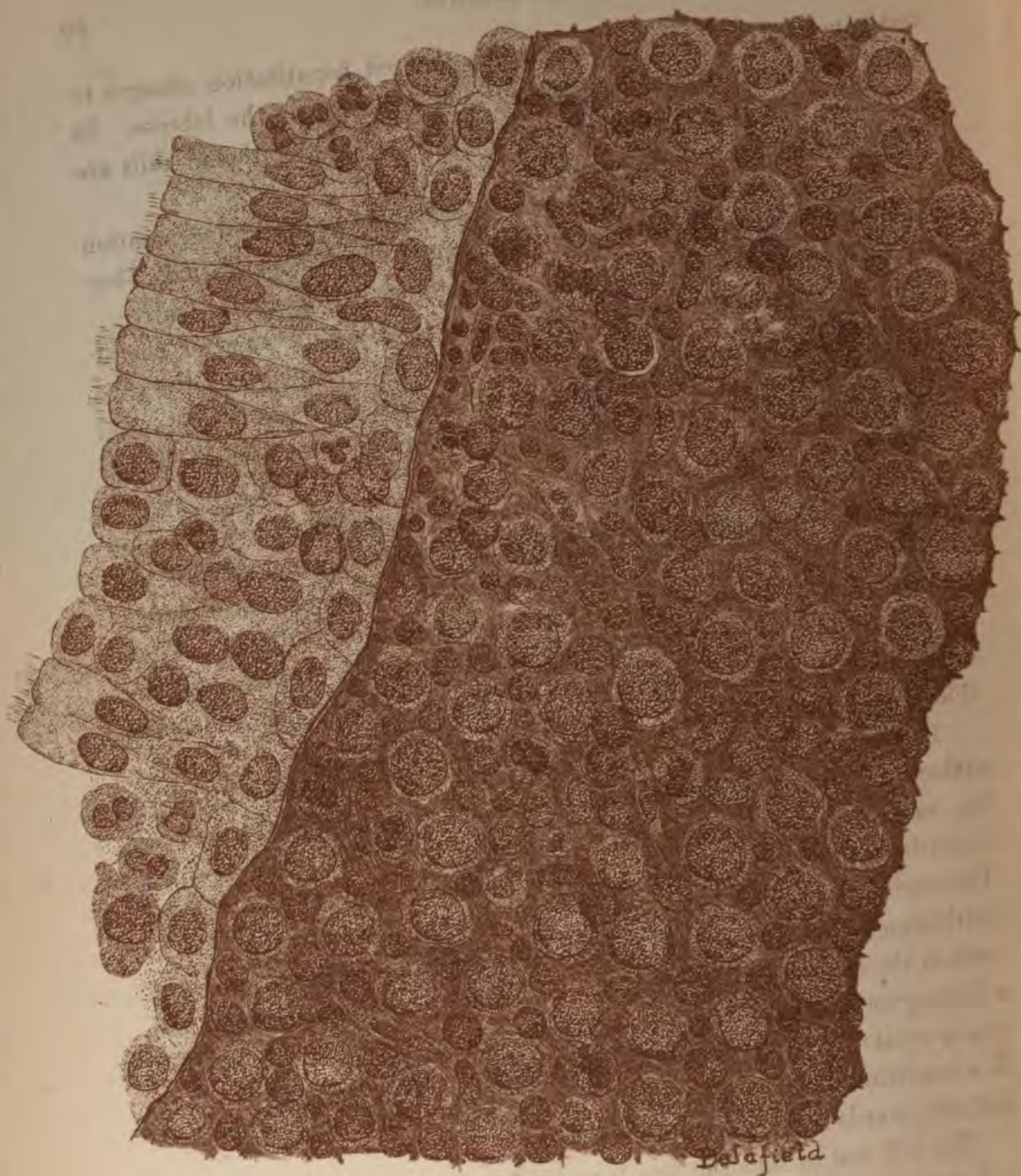


FIGURE No. 1

*Microscopic view of the
interior of the stem*



Delafield

PLATE XII.

*Part of the wall of a Bronchus.
Tubercular Pneumonia of Children.
magnified 850 diameters.*

red hepatization. If they live longer the red hepatization changes to gray, the change in color beginning in the centres of the lobules. In this condition of gray hepatization the pus and the epithelial cells are found swollen, granular, and fatty.

If resolution does not take place, the granular and fatty degeneration go on and the lobules pass into the condition of cheesy hepatization. They are then hard and yellow, the air-vesicles filled with granular matter.

It seems probable that resolution may take place even after cheesy hepatization is fully established. If it does not, however, after a time the cheesy lobules soften and cavities are formed, or there is interstitial pneumonia with production of new connective tissue, or both these processes go on together.

The condition of collapse of the lungs, or atelectasis, is one of a good deal of importance, particularly that variety which occurs as a complication of bronchitis. Larger or smaller bronchi become occluded, and the groups of air-vesicles which empty into them collapse. There can be no question of the frequency and importance of this condition, but I think that there is at present a tendency to call much atelectasis that is really pneumonia.

If we examine a portion of lung which is in the condition of atelectasis, without distending the air-vesicles, we find that the walls of the undilated vesicles give quite an irregular picture. They project inwards so that we no longer get the regular outline of the vesicles. The capillary vessels are large and may be distended with blood. The epithelium is more distinct, and there are a few large, granular epithelial cells in the cavities of the vesicles, with perhaps a few pus-globules and a little granular matter. That is all; there is no large number of either pus or epithelial cells. Sometimes, however, we find lobules of the lung in a condition which suggests the idea that there has been first collapse and afterwards inflammation.

The old test of inflating the lung to determine between atelectasis and pneumonia does not seem to be of very much value, for there are many degrees of pneumonia short of complete hepatization. There may be enough pneumonia to cause a partial filling up of the air-vesicles, but not sufficient to prevent their inflation.

The lobular pneumonia with the characters described above belongs properly to children under five years old. It is sometimes met with in older children, however, and I have seen it in persons between nineteen and twenty years old. It is said also that adults in whom bronchitis complicates measles may suffer from a lobular pneumonia like that of children.

V. Interstitial Pneumonia.

This term is applied to a chronic form of inflammation involving the fibrous framework of the lung and resulting in the production of permanent new connective tissue. The new tissue thus produced is dense, contains comparatively few cells, and is often pigmented. It is usually arranged in the form of nodules, of bands, or of irregular patches. The walls of the air-vesicles, of the bronchi, and of the blood-vesicles may all be involved in such an inflammatory process.

The examples of the disease, which have come under my observation, have been in connection with emphysema and chronic bronchitis, with anthracosis, with pleurisy, and with chronic phthisis.

In emphysematous lungs there are sometimes numerous small, hard nodules, sometimes bands and patches of fibrous tissue, which seem to have no relationship with phthisis or tuberculosis, but to be simply the results of an interstitial inflammation.

The inhalation of coal dust and stone dust may excite an interstitial pneumonia, but I have not seen enough cases of these lesions to speak of them with confidence.

Extensive old pleuritic adhesions with thickening of the pulmonary pleura are often accompanied by the production of bands of fibrous tissue extending from the pleura inwards into the lungs. The extreme examples of this lesion, when the entire lung is converted into fibrous tissue and the bronchi dilated, have not fallen under my observation.

The interstitial pneumonia of phthisis will be described farther on.

VI. The Pneumonia produced by pressure on the Trachea and Bronchi.

If an aneurism presses upon the trachea or upon the right or left bronchus it usually gives rise to a catarrhal inflammation of the trachea

and bronchi. In a certain number of cases there are also characteristic changes in the lungs. If the trachea is compressed by the aneurism, both lungs may be affected; if one bronchus is compressed, then the lung belonging to that bronchus will be alone affected.

The appearance of the lungs is something like that of the lungs in phthisis. The lung is thickly studded with hepatized lobules from the size of a pea to that of a chestnut. Some of the lobules look like red hepatization, more are gray or cheesy, some are softened and converted into cavities containing puriform fluid. If the aneurism has partly opened into the trachea or bronchus so that small quantities of blood are inspired, then some of the hepatized lobules are partly filled with blood.

Minute examination, however, shows the lesions to be different from those of phthisis. There seems to be a combination of three different processes: (1) an intra-alveolar pneumonia; (2) collapse of groups of air-vesicles; (3) an interstitial pneumonia. I have not yet seen enough cases of this lesion to speak of it with certainty, but my belief is that the process is first a bronchitis, then a plugging of the bronchi with pus and mucus, then a collapse of groups of air-vesicles, after this an interstitial pneumonia involving the walls of the collapsed vesicles and an intra-alveolar pneumonia involving the interior of the air-vesicles which surround the collapsed lobules, and resulting in the filling of these vesicles with epithelium and pus.

ACUTE MILIARY TUBERCULOSIS.

ALTHOUGH all are not agreed as to the causes and nature of miliary tubercles, yet it must be allowed that important advances have been made in our knowledge of their anatomy.

From the time that Virchow described them as organized tissue, composed of cells, and not as amorphous masses with shrivelled nuclei, the study of their minute anatomy has been steadily pursued. Virchow compared them to lymphatic glands, laying especial stress on the cells, which he described as small round cells with a small cell body, and large nucleus. Wagner and others have called attention to the existence of a basement substance in which the cells are imbedded, and which resembles the basement substance of connective tissue, and have spoken of tubercles as composed of adenoid tissue.

Schüppel and others have shown that giant-cells are frequently present, and that many of the apparently round cells really have a polygonal cell body of some size, and resemble connective-tissue cells rather than the cells of lymphatic glands.

Charcot and others have shown that it is possible that the giant-cells and other cells may be developed from the epithelium of the air-vesicles, and that the giant-cells are concerned in forming the basement substance of the tubercle.

The tendency has been more and more to establish a definite anat-

omy for tubercle, although many still call any little nodule formed of round cells and partly cheesy, a miliary tubercle. There is also a good deal of confusion as to whether miliary tubercles should be regarded as new growths, or as products of inflammation.

So far as the gross appearance of miliary tubercles is concerned, almost every one has followed the description of Laennec.

"The tubercles," says Laennec, "resemble small grains; they are of a gray color, and semi-transparent, sometimes even transparent and colorless, and of a consistence somewhat less than that of cartilage. Their size varies from that of a millet to that of a hemp seed. Their shape, roundish at first sight, is found on inspection with a lens to be less regular. They adhere intimately to the pulmonary substance, and a number of them may be united so as to form a group. After a certain time a yellowish, opaque speck appears in the centre of each tubercle."

Miliary tubercles of this character have been observed in almost every part of the body. They may be developed in the course of a few days and be accompanied by acute constitutional symptoms; or they are formed slowly and remain during months and years. We speak, therefore, of acute tuberculosis and of chronic tuberculosis. We will consider, first, the miliary tubercles, which are produced in a short space of time.

In the *lungs* miliary tubercles are usually numerous, and are found both in the parenchyma of the lung and the pulmonary pleura.

The lung tissue between the tubercles may appear to the naked eye normal. Very frequently it is congested, or partly hepatized. Minute examination always, so far as I know, shows some changes in the air-vesicles, even when the lung tissue appears normal to the naked eye. The change in the air-vesicles consists in a production, within their cavities, of large, polygonal (epithelial) cells, of pus-cells, of fibrine, and of red blood-globules. The amount and proportion of these inflammatory products vary in the different cases.

The bronchi also usually show changes; their mucous membrane is congested and coated with mucus.

The miliary tubercles are scattered singly through the lungs, or are



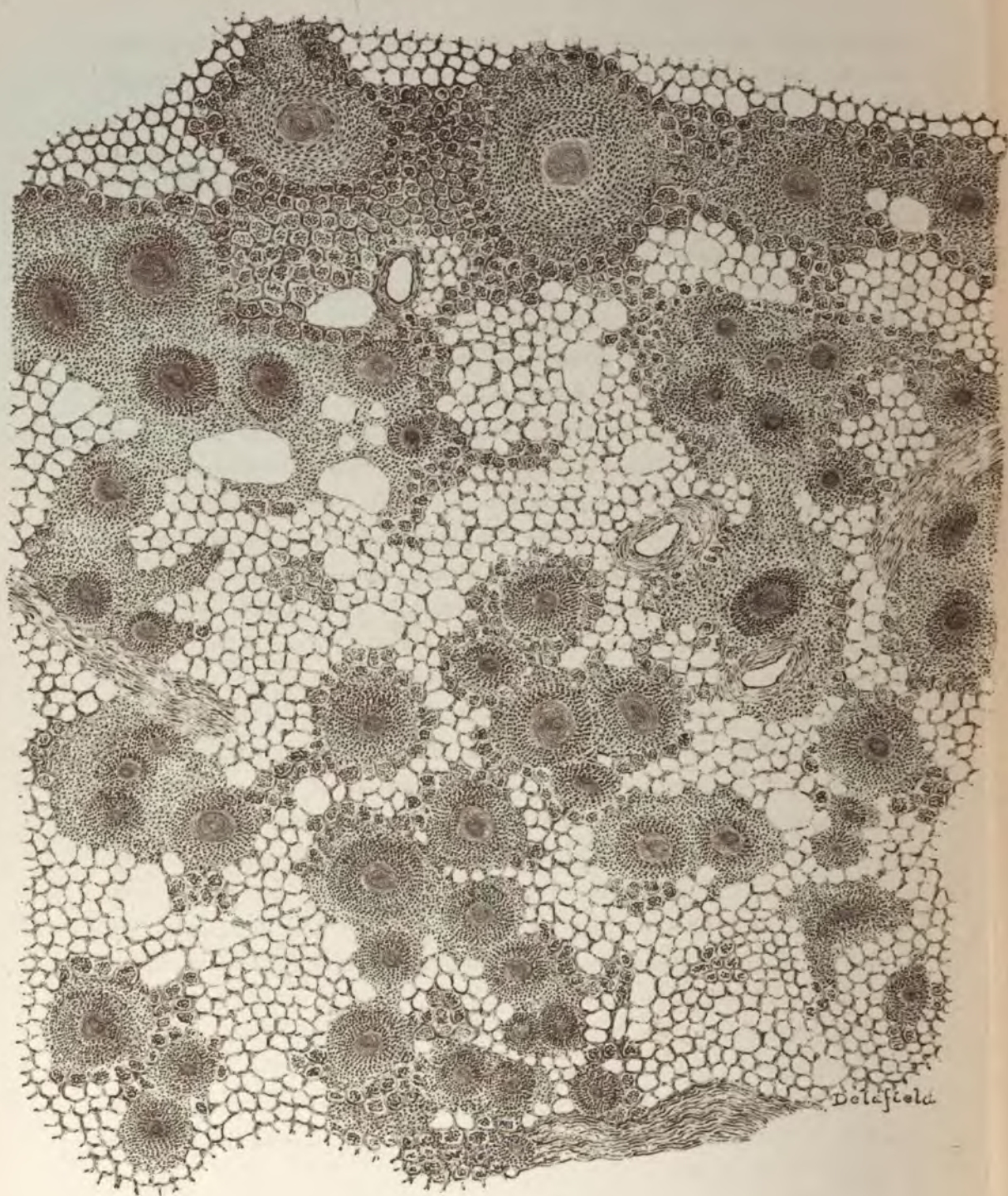


PLATE XLIII.

*Miliary Tubercles;
magnified 22 diameters.*

aggregated in groups, or situated close to each other and joined together by more diffuse infiltrations of similar tissue. They may be separated from each other by considerable intervals of lung tissue, or be so close together that the lung is rendered almost solid by them. There is much variety in the size of the tubercles; most of them are about the size of a pin's head, but some are so small they can hardly be seen with the naked eye, and occasionally they are as large as peas. Their consistence may be as hard as cartilage, or as soft as coagulated fibrine, and between these extremes are all gradations. In color some are gray and semi-translucent, some are white, some are gray or white with yellow centres, some are entirely yellow. They all have a tendency to pass into the condition of cheesy degeneration, but the degree in which this tendency is developed varies in different lungs. In some cases a large number of the tubercles escape degeneration altogether; in some only their centres are cheesy; in some the larger number are completely cheesy. The cheesy matter may be hard and dense, or soft and friable. The extent of cheesy degeneration has generally a relation to the age of the tubercles: the older the tubercles, the more cheesy degeneration. This rule, however, is not without exceptions.

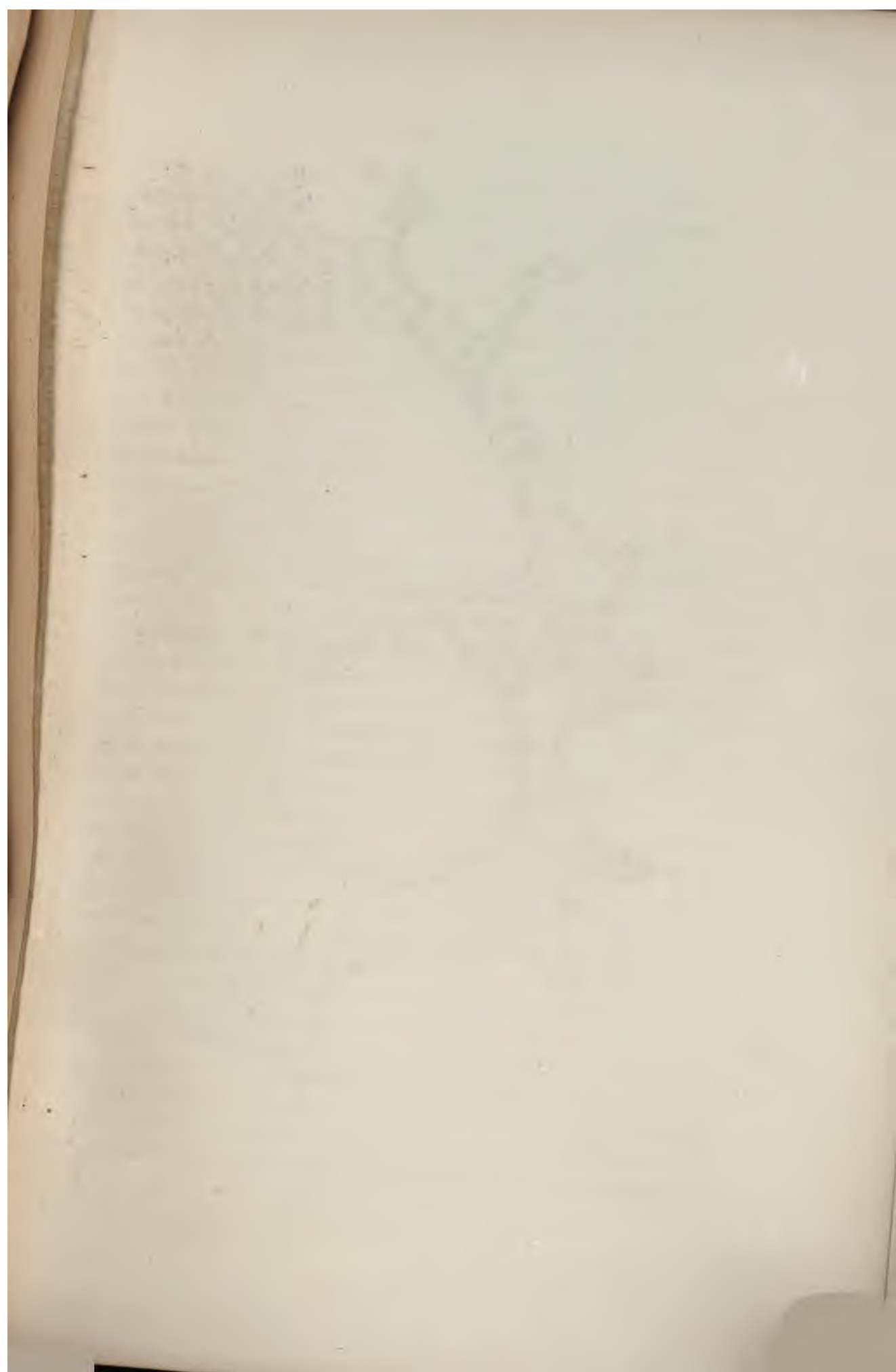
If we look at the tubercles with a low magnifying power we see that some are of spheroidal shape, some look like irregular infiltrations, and some are joined together so as to form masses of some size. The appearances vary with the number of the tubercles, their arrangement, and the amount of consolidation that has taken place in the intervening lung. Plate XLII. represents a section of lung magnified twenty-two diameters. The tubercles are numerous and close together, many are united to neighboring tubercles by consolidated lung, all have undergone cheesy degeneration at their centres.

When we look at the individual miliary tubercles with higher magnifying powers, and compare them as they exist in different lungs, it soon becomes evident that they are not all of the same structure, and that it is necessary to distinguish four principal varieties of miliary tubercles in the lungs.

(1.) Miliary tubercles composed entirely of amorphous granular matter, with a few shrunken cells, and an external zone of pus-cells.

These tubercles cannot be said to have a definite anatomical structure. Many of them look as if they were the results of cheesy degeneration of pre-existing anatomical elements, the walls of the air-vesicles sharing in the degenerative process. In others, however, the walls of the air-vesicles are intact, while their cavities are filled with amorphous granular matter. At the first glance it may be thought that this variety of miliary tubercles hardly needs any separate mention; that it is merely a question of the extent of the degenerative process; that these tubercles have the same structure as others, but are further advanced in degeneration. In the larger number of all miliary tubercles it is indeed the rule for the centre of the tubercle to undergo cheesy degeneration and be converted into amorphous granular matter, and in many lungs we can see the different steps of the degenerative process and the conversion of more and more of the original anatomical elements into granular matter. But the miliary tubercles of which I am now speaking present no such gradations. In the entire lung all the tubercles are simply masses of granular matter with pus-cells at their peripheries. The only difference between the smaller and larger tubercles is that in the smaller ones the walls of the air-vesicles are still visible, and their blood-vessels can be injected; in the larger, the walls of the vesicles are destroyed. I am very far from saying that in these tubercles there have not been anatomical elements first produced, which have afterward degenerated. But I think that the earliness and completeness of the transformation of these tubercles and the absence of any visible intermediate stages of degeneration entitle them to separate consideration. A possible explanation seems to be that in these cases there is no formation of regular anatomical elements, but that from the very first amorphous inflammatory products are formed which rapidly degenerate and involve with them in their destruction the walls of the air-vesicles. Miliary tubercles of this same indeterminate character are also not uncommon in the peritoneum.

(2.) Miliary tubercles composed principally of new tissue, which apparently replaces the parenchyma of the lung, while at the periphery of this nodule of new tissue are air-vesicles filled with cells. Plate XLV. represents a miliary tubercle of this kind. In the right of the field





PLA

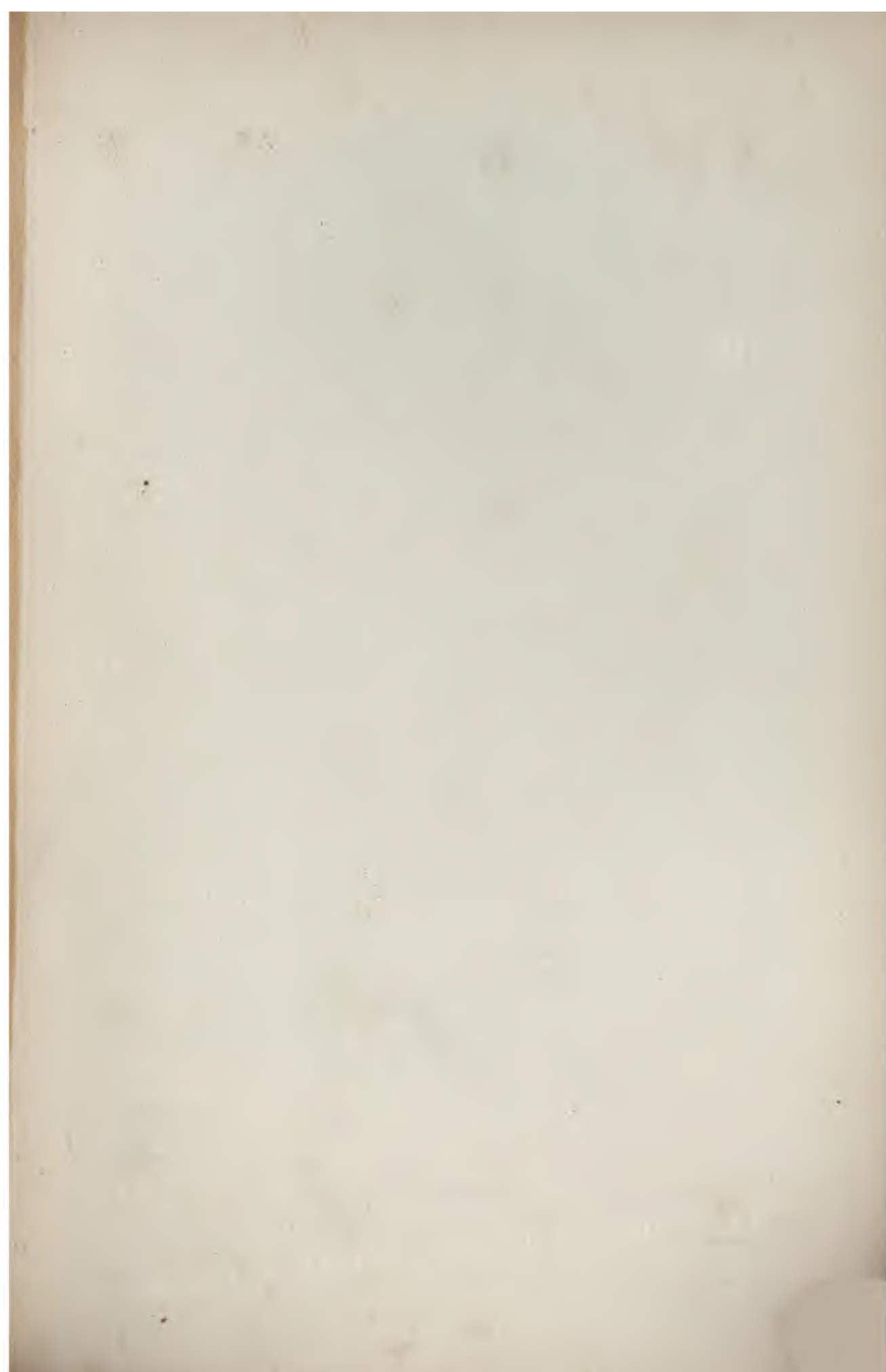
*A single
magnif*



XLV.

*Tubercle,
diameters.*





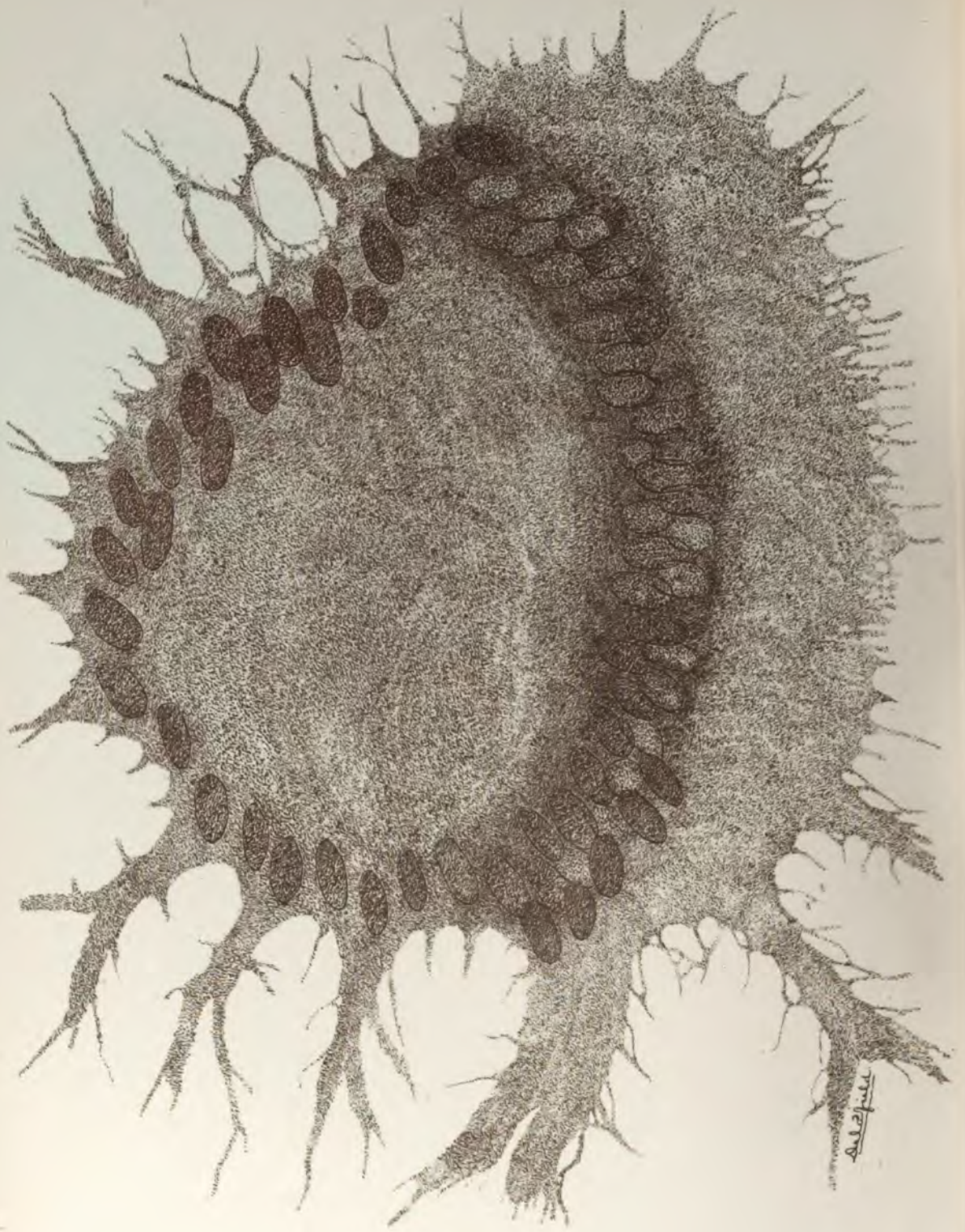


PLATE XLVIII.

*A Giant cell;
magnified 1500 diameters.*

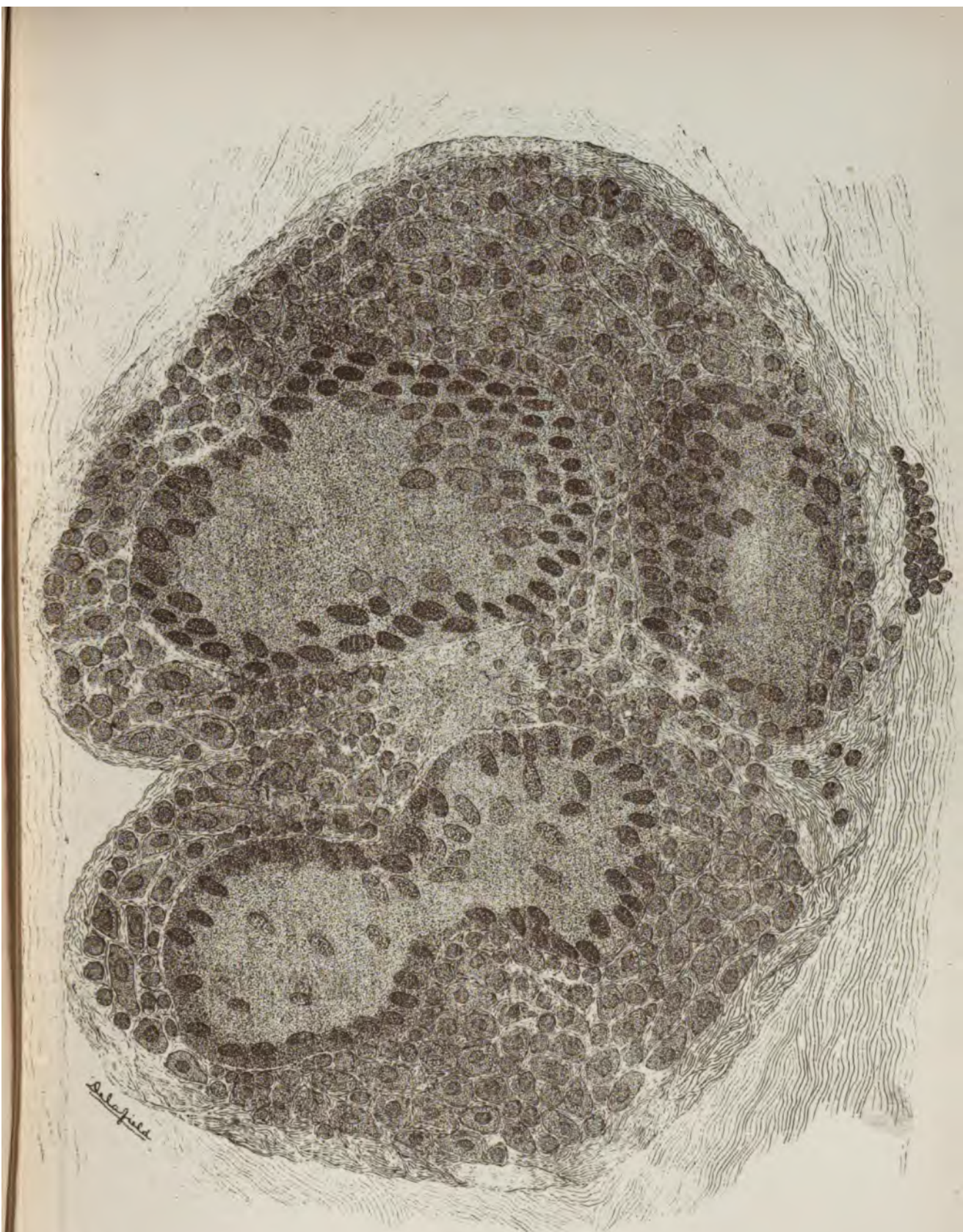
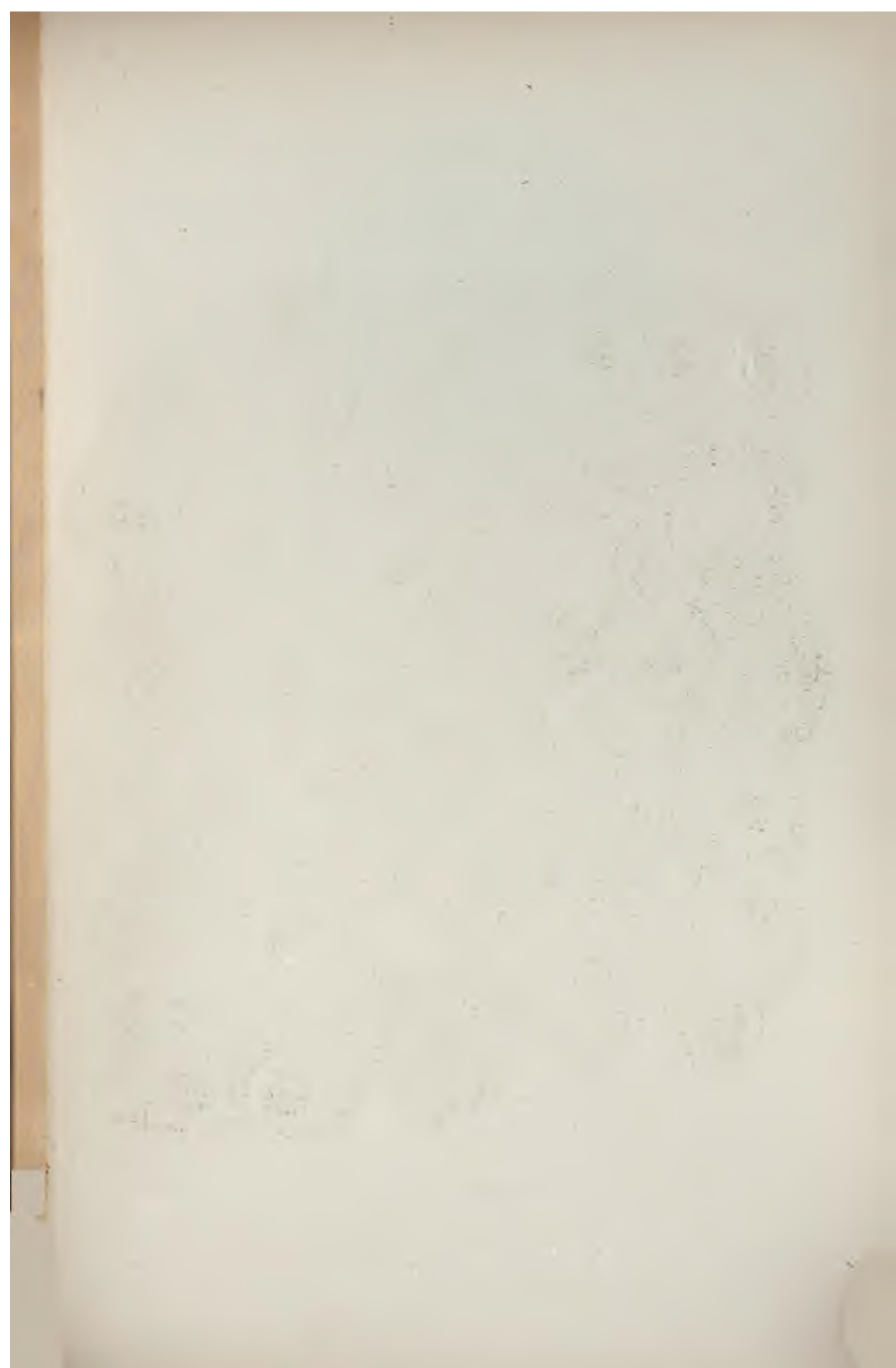


PLATE XLIX.

*Tubercle, A Tubercle Granulum, Lupus of the Face,
magnified 750 diameters.*





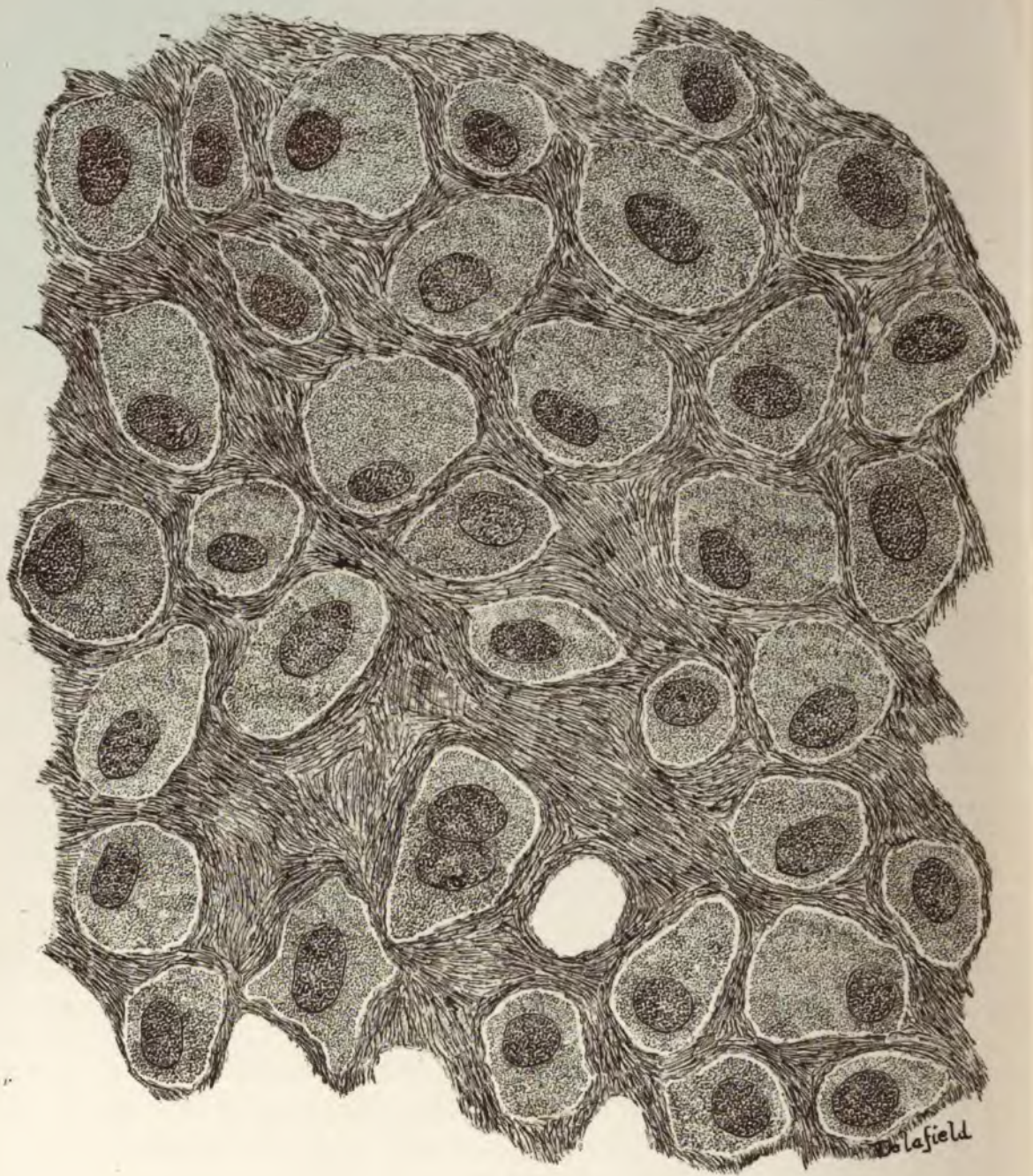


PLATE XLIV.

*Part of a tubercle granulum;
magnified 1500 diameters.*

is a blood-vessel, to the left of this is a mass of solid tissue occupying most of the field, while at the edges of this mass are air-vesicles, of which the walls are covered with large (epithelial) cells. In one air-vesicle an invasion of the solid tissue into the cavity of the vesicle seems to have taken place, although the outline of the wall of the vesicle is preserved. The solid tissue is composed of elements, having the same general appearance but arranged in two different ways. Part of the tissue is arranged so as to form two irregularly spherical bodies, and the rest as a diffuse infiltration around and between these two bodies. The spherical bodies we may call, for convenience, "tubercle granula;" the diffuse tissue, "diffuse tubercle." When we examine the tubercle granula more carefully, we see that the centre of each is occupied by a large mass of protoplasm filled with nuclei. If we isolate one of these masses of protoplasm, or giant cells, we see that it is of such a form as is represented in Plate XLVIII., and that its branching processes are apparently continuous with the basement substance situated between the surrounding cells. The rest of each tubercle granulum is composed of cells of irregularly polygonal shape imbedded in a basement substance as in Plates XLIV., XXIX., and XLIX. The bodies of the cells are very delicate and easily escape observation unless carefully prepared; the nuclei, on the contrary, are readily seen. Besides these polygonal cells, there may be round cells looking like pus globules. The basement substance between the cells varies in its character in different tubercles; it may look like the basement substance of reticulated connective tissue, or it may be a delicate, finely granular material requiring careful demonstration, and between these two extremes we see many gradations. The basement substance sometimes, but not always, appears to be continuous with the processes of the central giant-cell. The giant-cells are indeed often entirely absent from the tubercle granula, and these latter are then composed only of the basement substance, polygonal cells and round cells. When the giant-cells are present, they may be situated in any part of the tubercle granulum, not necessarily in its centre; there may be two or three such giant-cells in a single granulum. The diffuse tubercle has the same structure as the tubercle granula—polygonal cells and small

round cells imbedded in a basement substance. See Plate XLVI. The basement substance, however, is better marked than in the tubercle granula, the bodies of the polygonal cells are less delicate, the round cells are more numerous, and the giant-cells are less constantly present.

This variety of miliary tubercle may become the seat of cheesy degeneration. I think, however, that the extent and constancy of cheesy degeneration of all miliary tubercles has been somewhat exaggerated by authors. Such degeneration is common enough, but there are very many miliary tubercles of which all the anatomical elements are well preserved. Even in those tubercles, which look opaque at their centres, there may be no destruction of tissue.

It is very difficult to be certain of the earlier stages of this form of tubercle. Some authors are disposed to believe that the process begins in the cavities of the air-vesicles with a subsequent growth of new tissue in their walls. It is said that there is first a growth of cells within the air-vesicle, a transformation of some of these cells into one or more giant-cells, a formation of basement substance from the body of the giant-cell, and then an infiltration and thickening of the wall of the air-vesicle. If this explanation were true, then each tubercle granulum is a single air-vesicle, or bronchiole, with thickened walls and filled cavity. This explanation is plausible enough, but it is difficult to really demonstrate the different steps of the process. The mere shape of the tubercle granula does not at all show that they are altered air-vesicles, for they are of the same shape in whatever part of the body they occur. See Plate XVIII.

(3.) Miliary tubercles composed partly of solid tissue, partly of air-vesicles filled with tubercle tissue, partly of air-vesicles filled with epithelium, pus, and fibrine. Plate XLIII. represents a section of a single miliary tubercle of this kind. It will be seen that in the upper and left hand portion of the plate, and extending to its centre, is a group of five tubercle granula surrounded by diffuse tubercle. In these granula the cells are larger than in those in Plate XLV., and there are no giant-cells. In the granulum in the centre of the drawing the tissue is broken down and softened, otherwise the structure of the granula and of the diffuse tubercle is the same as of those in Plate XLV. In

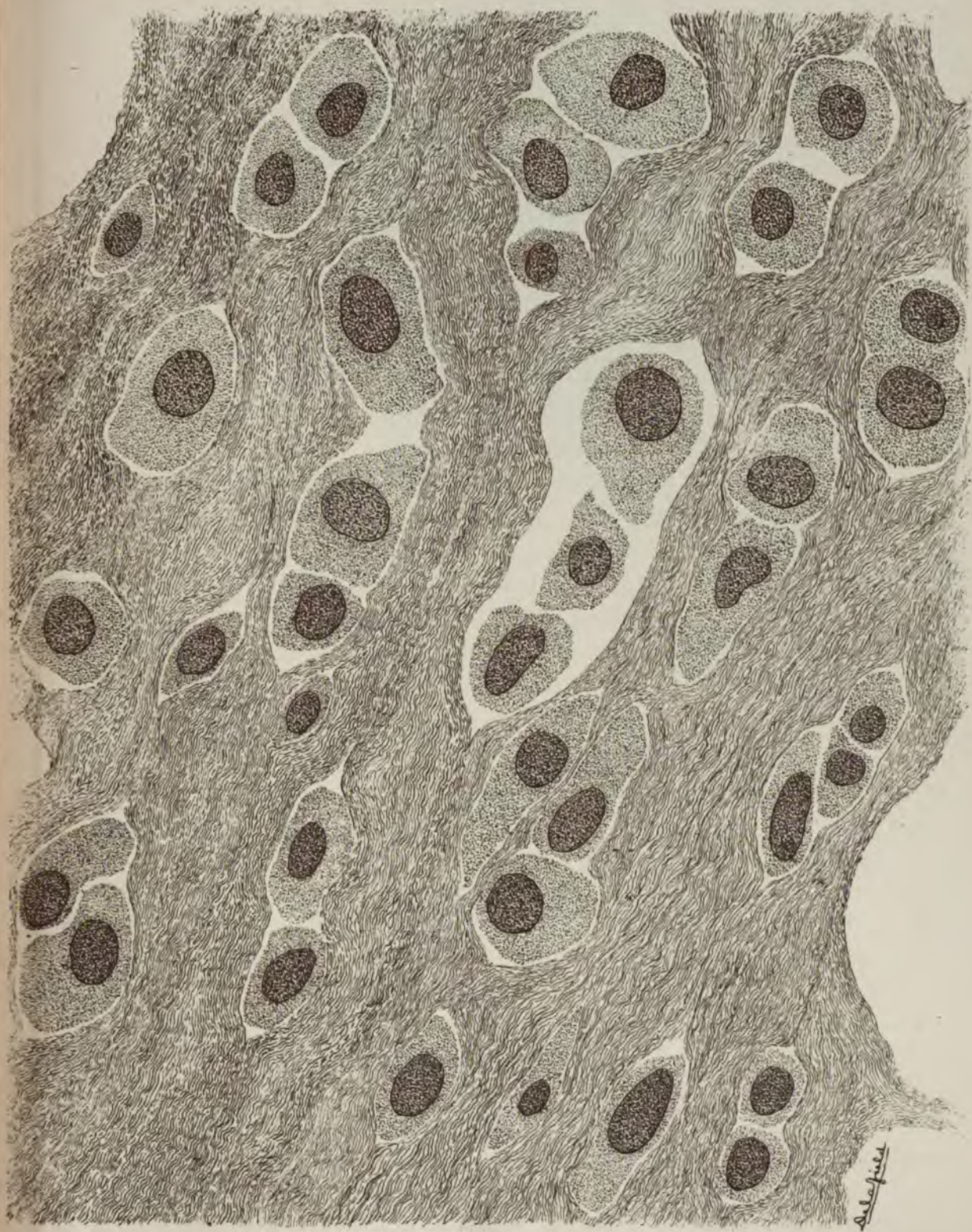
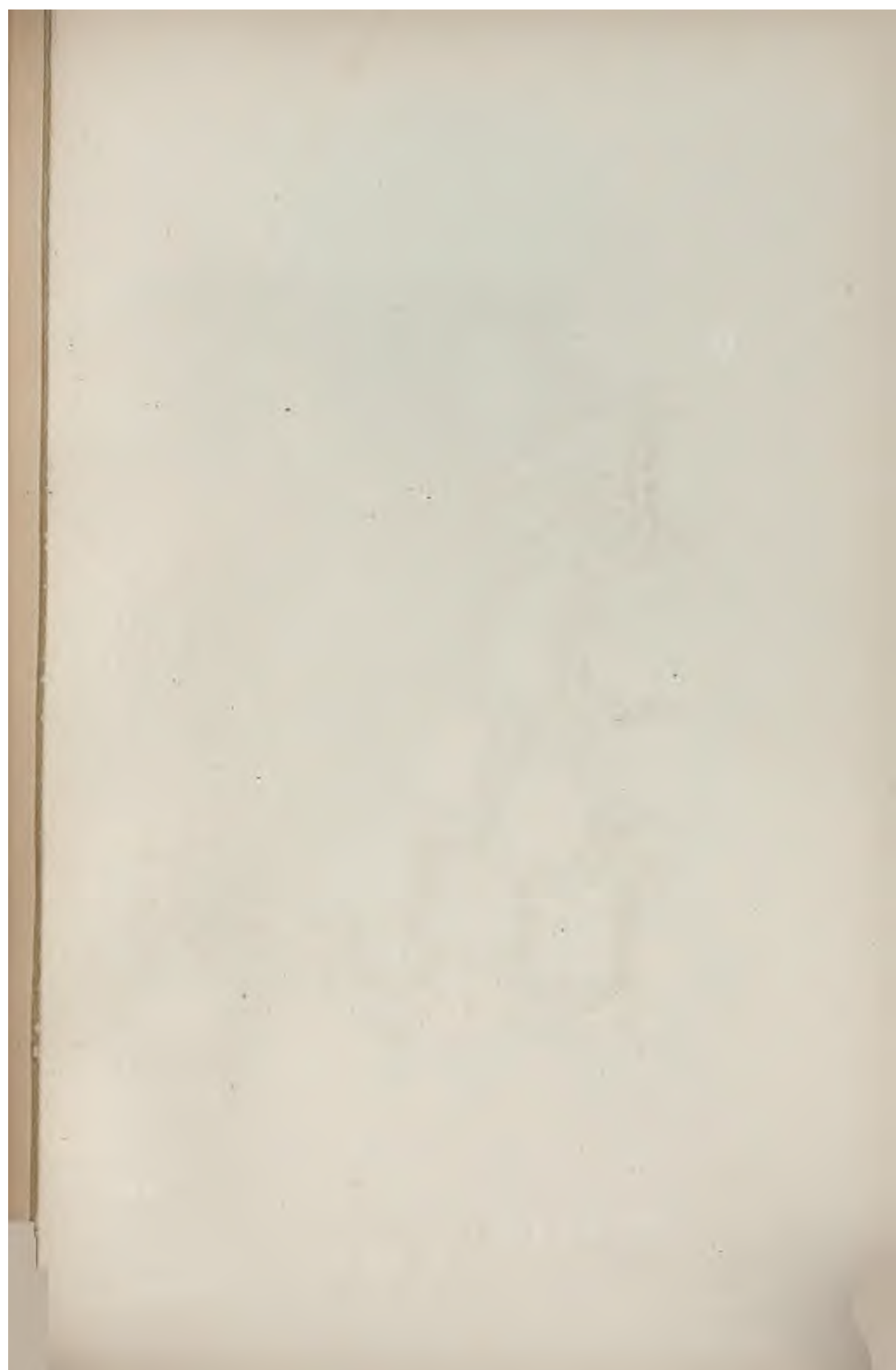
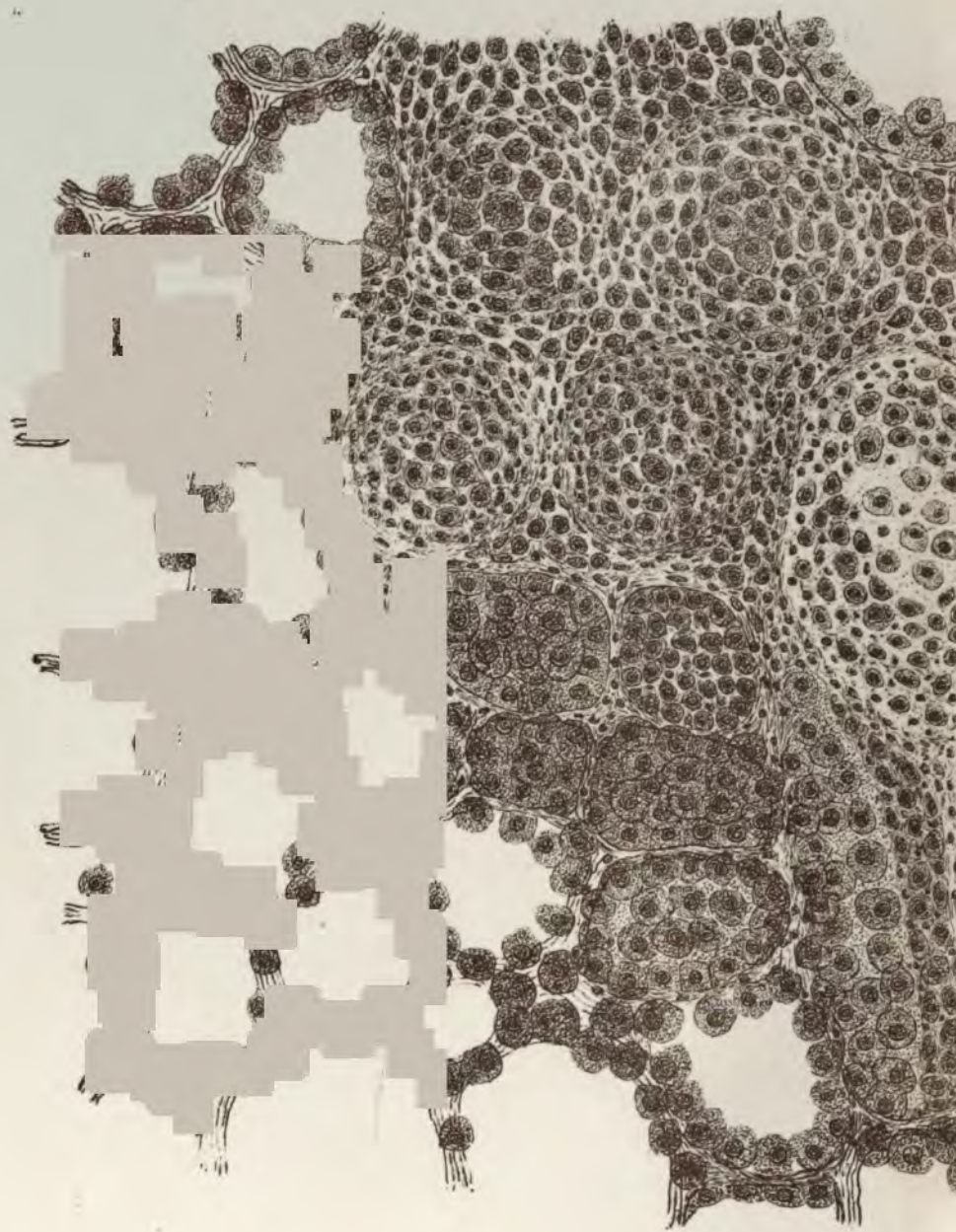


PLATE XLVI.
Diffuse Tubercle;
magnified 1500 diameters.

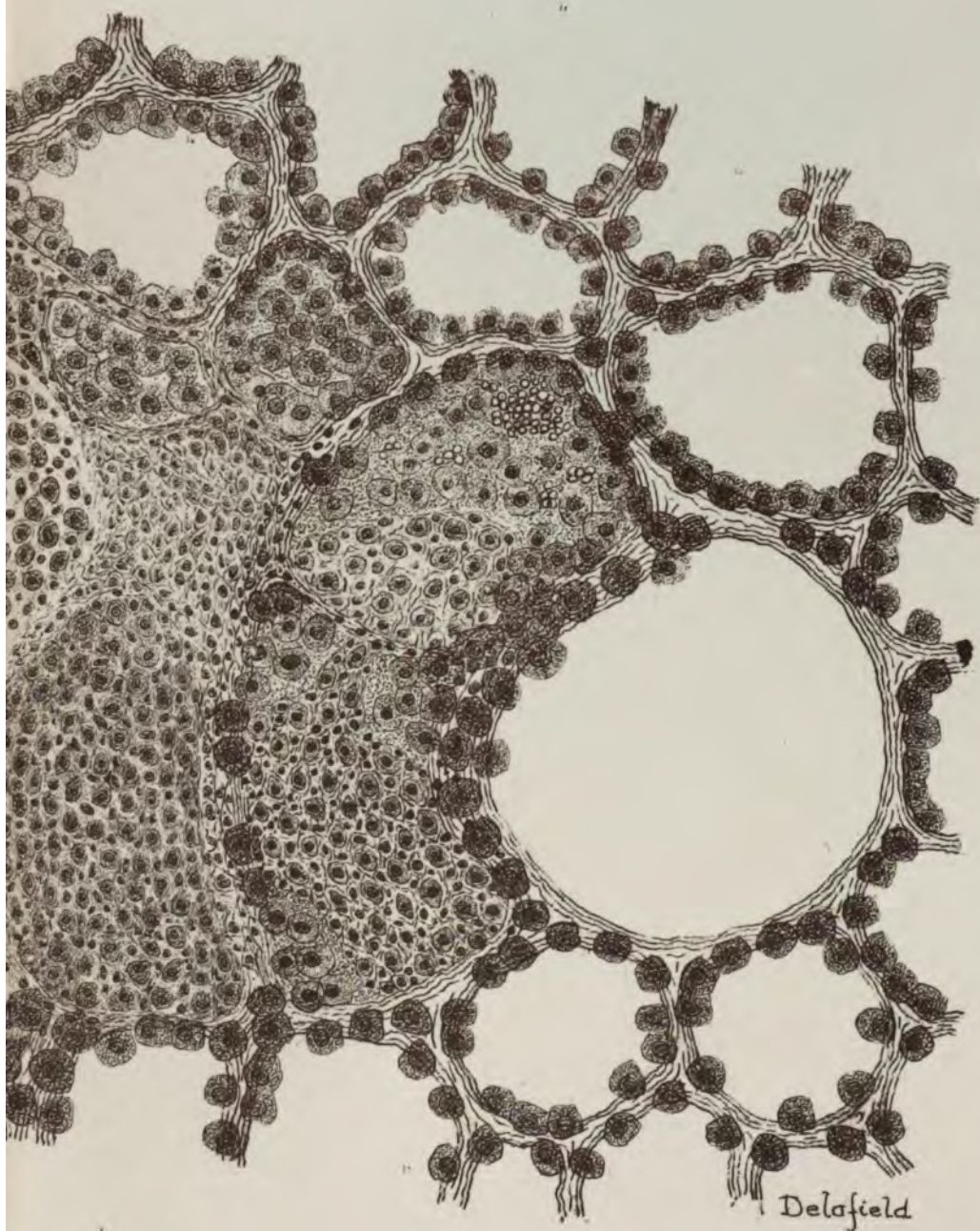






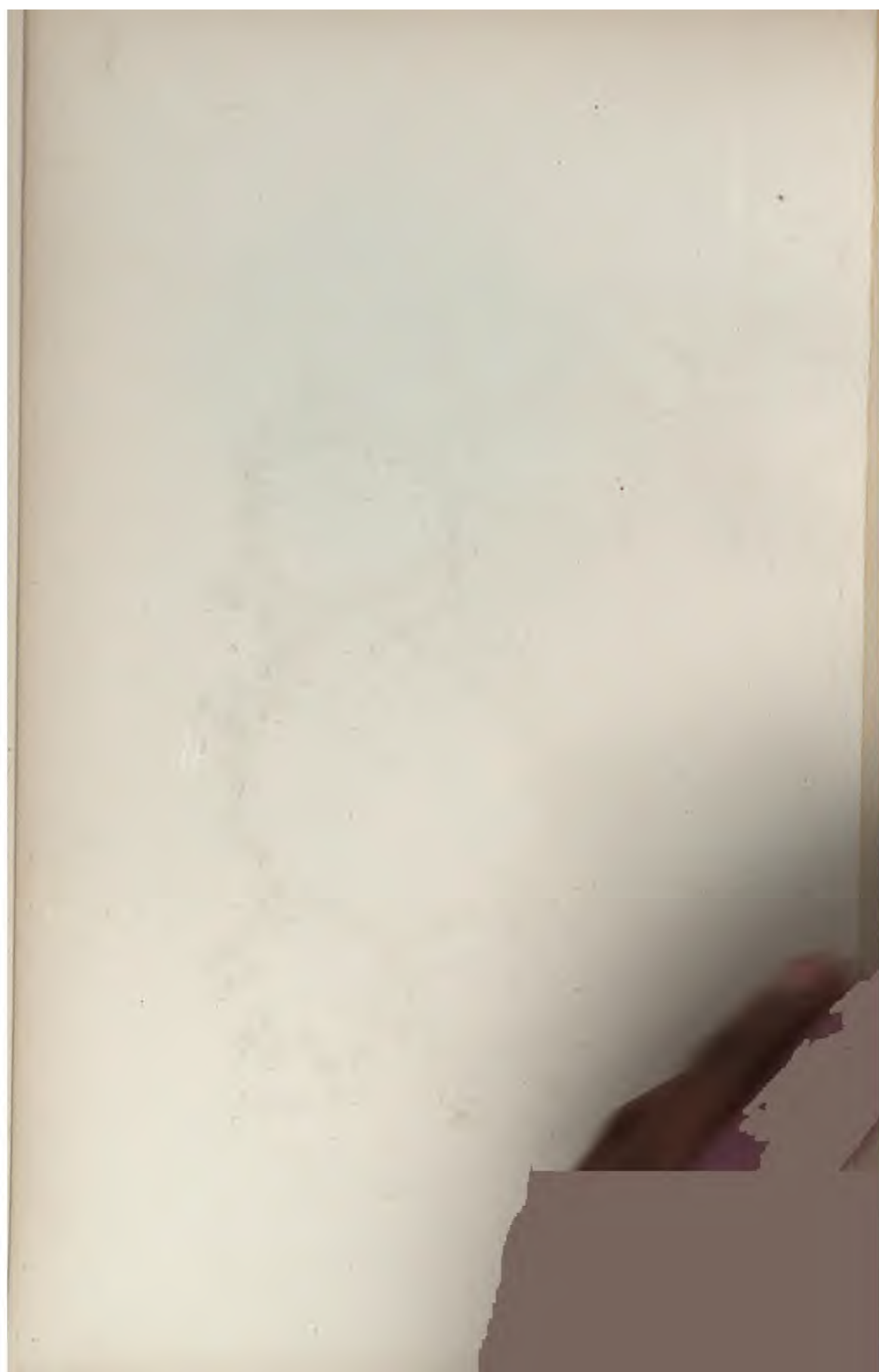
PLATE

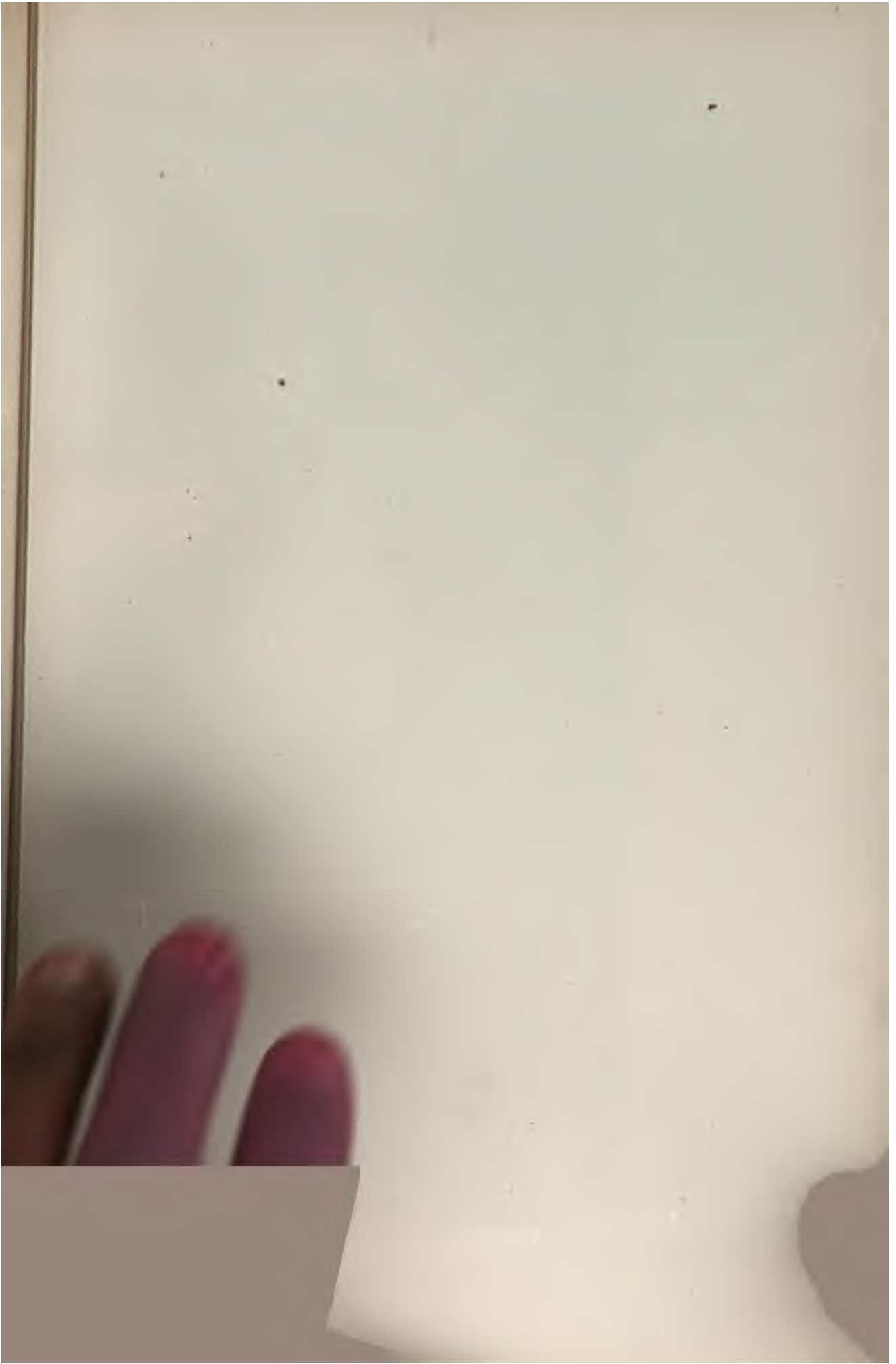
*A military
magnified 30*



III.

*Tubercle
cavitous.*

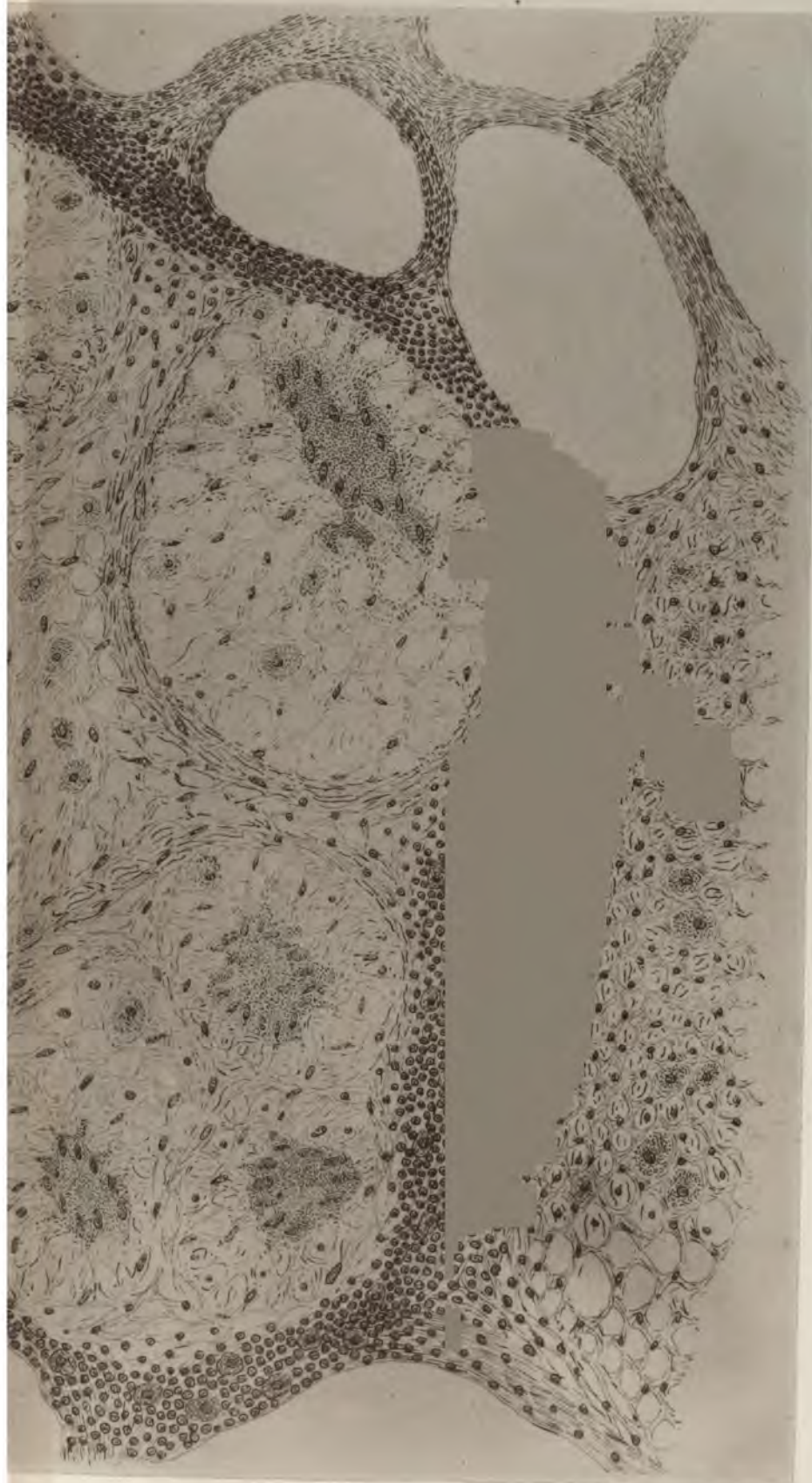






ARTOTYPE

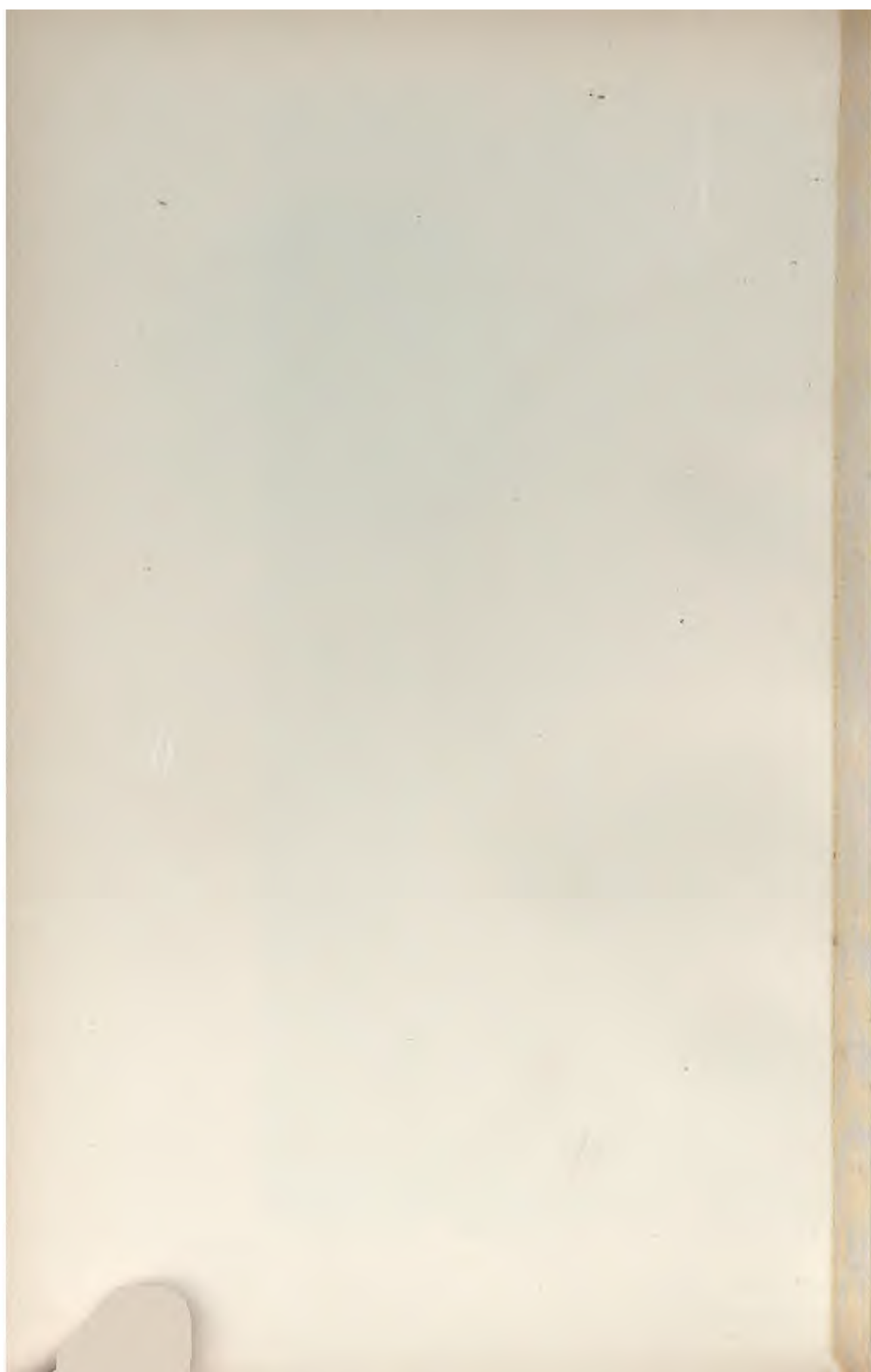
Plate XLIII
ACUTE MILIARY

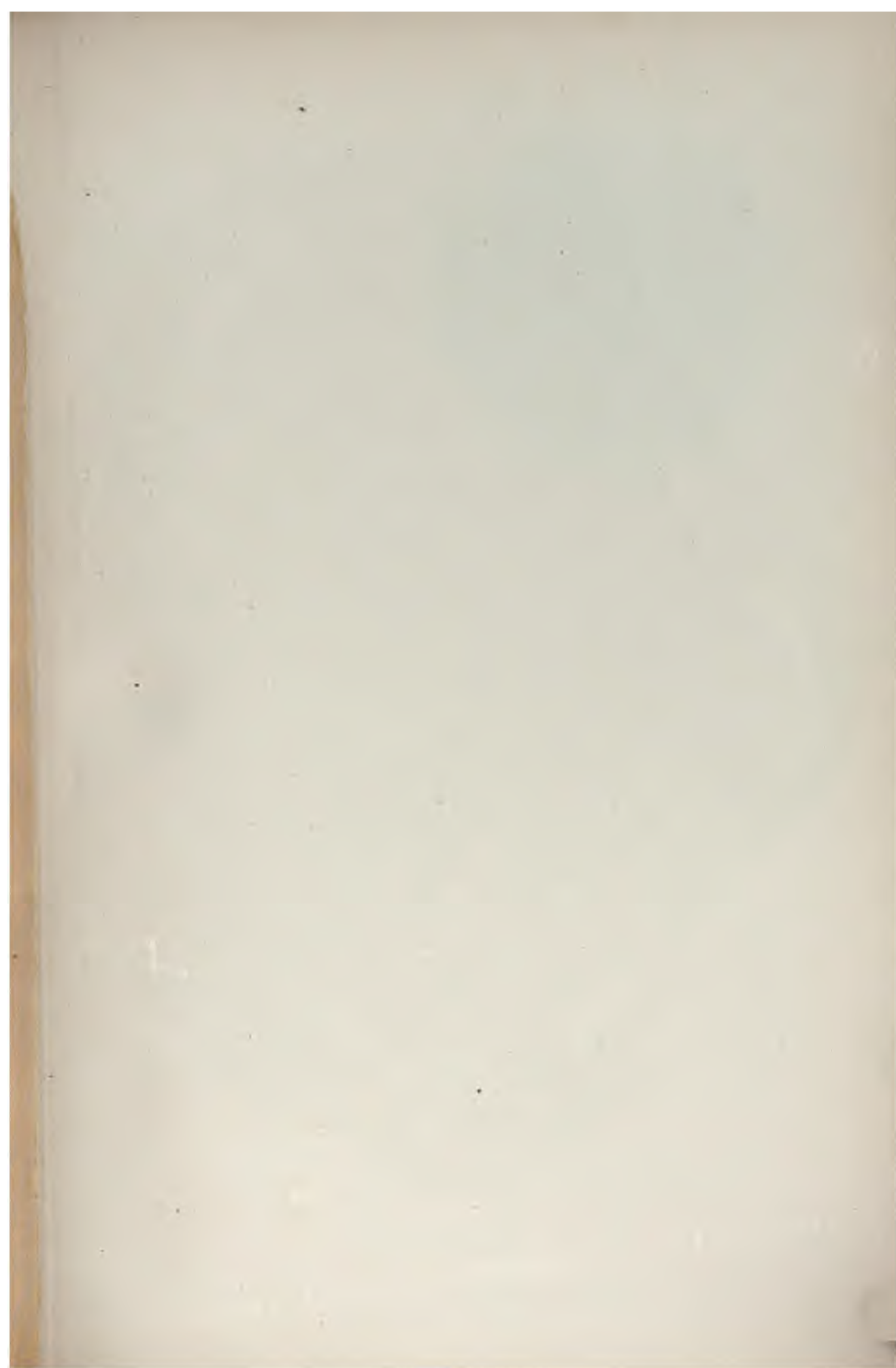


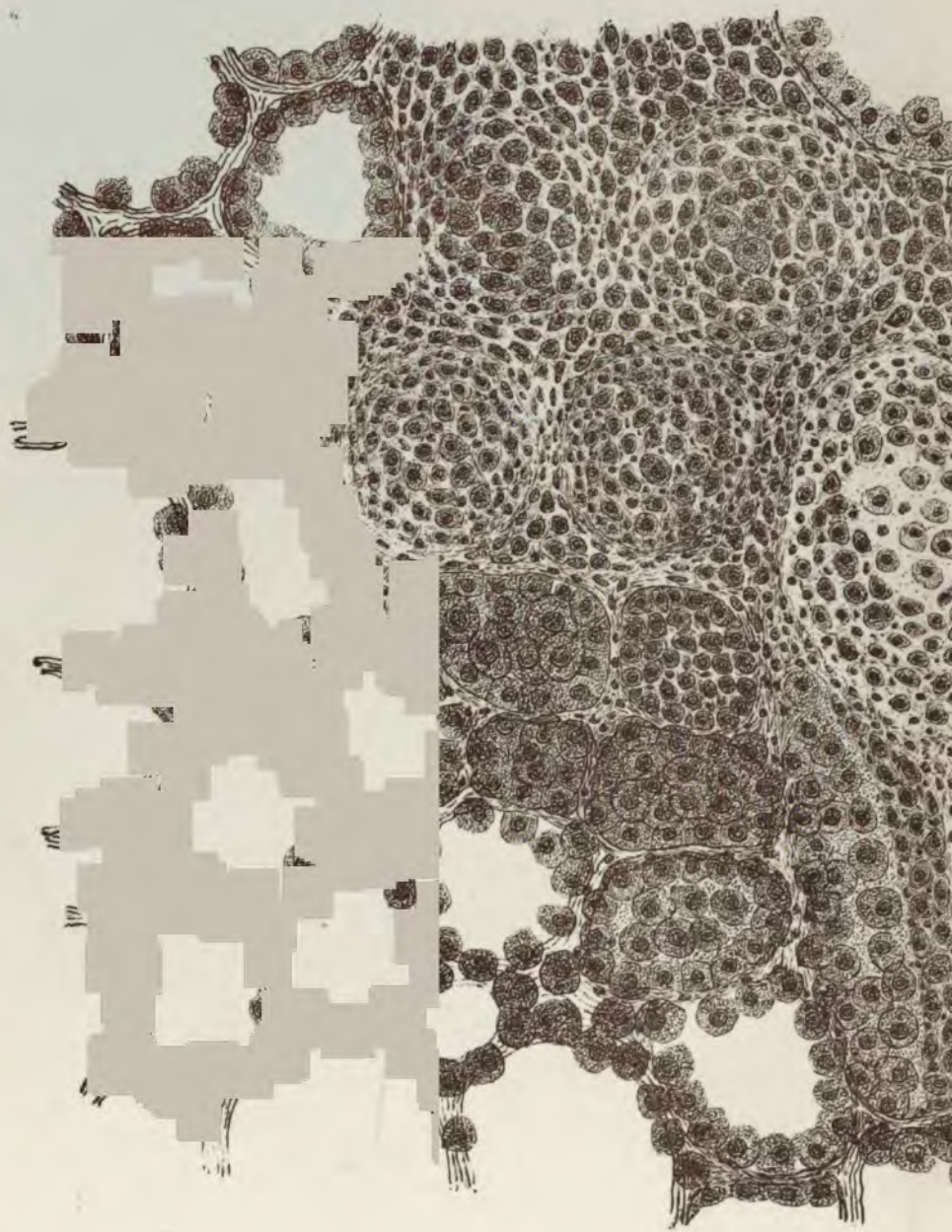
E. HERRMANN, A. V.

I. No 2. x 300

TUBERCULOSIS.

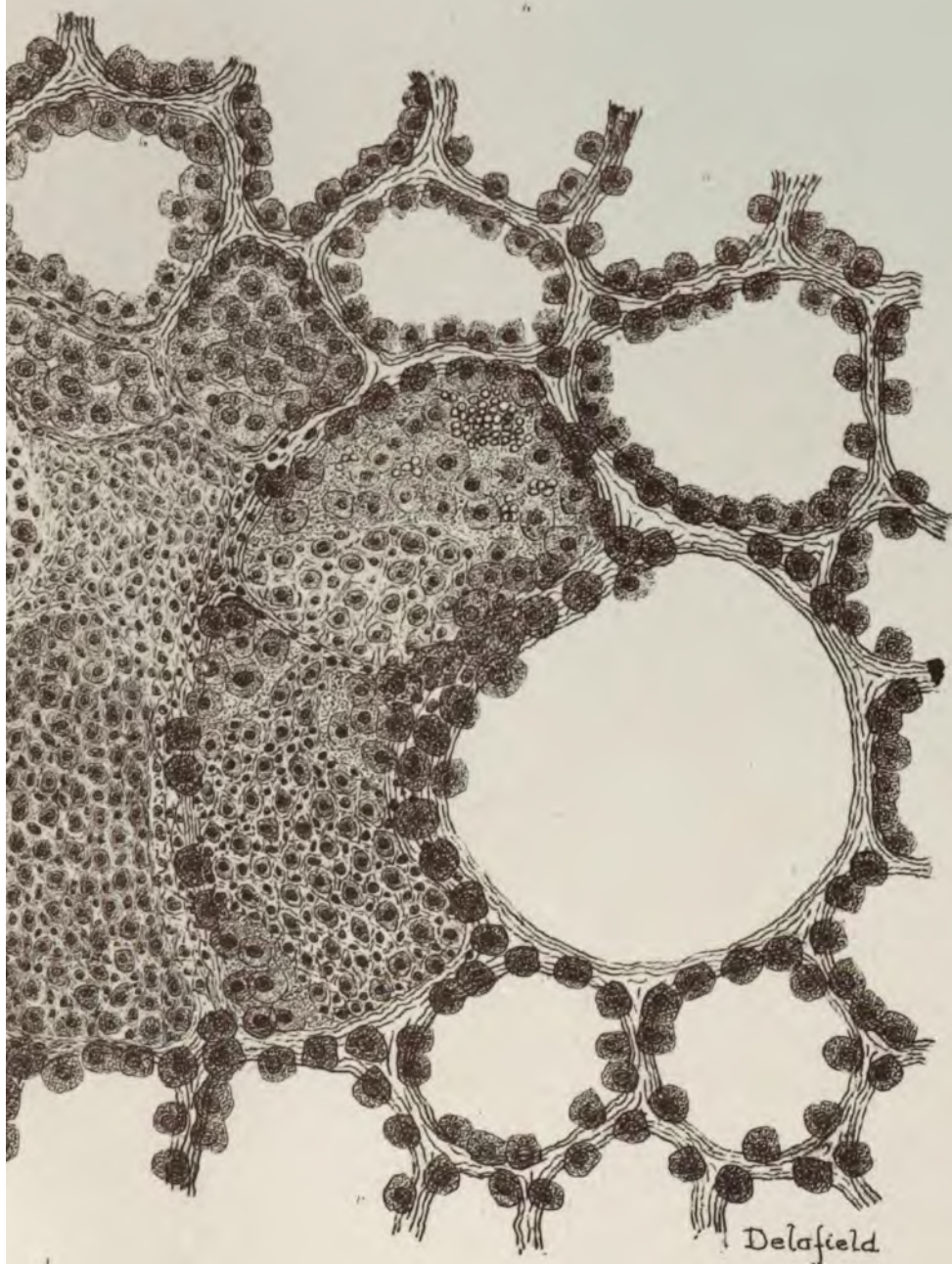






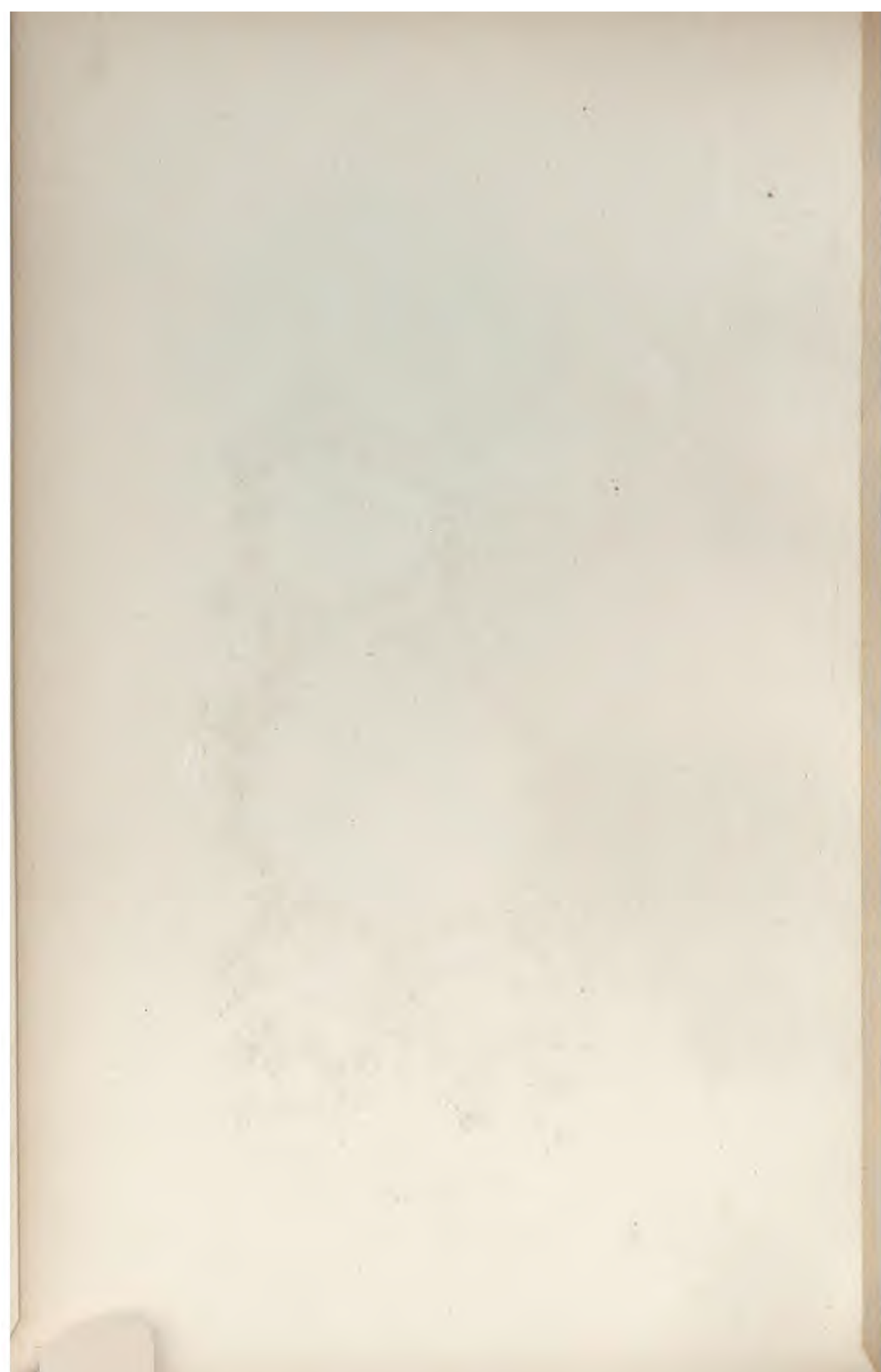
PLATE

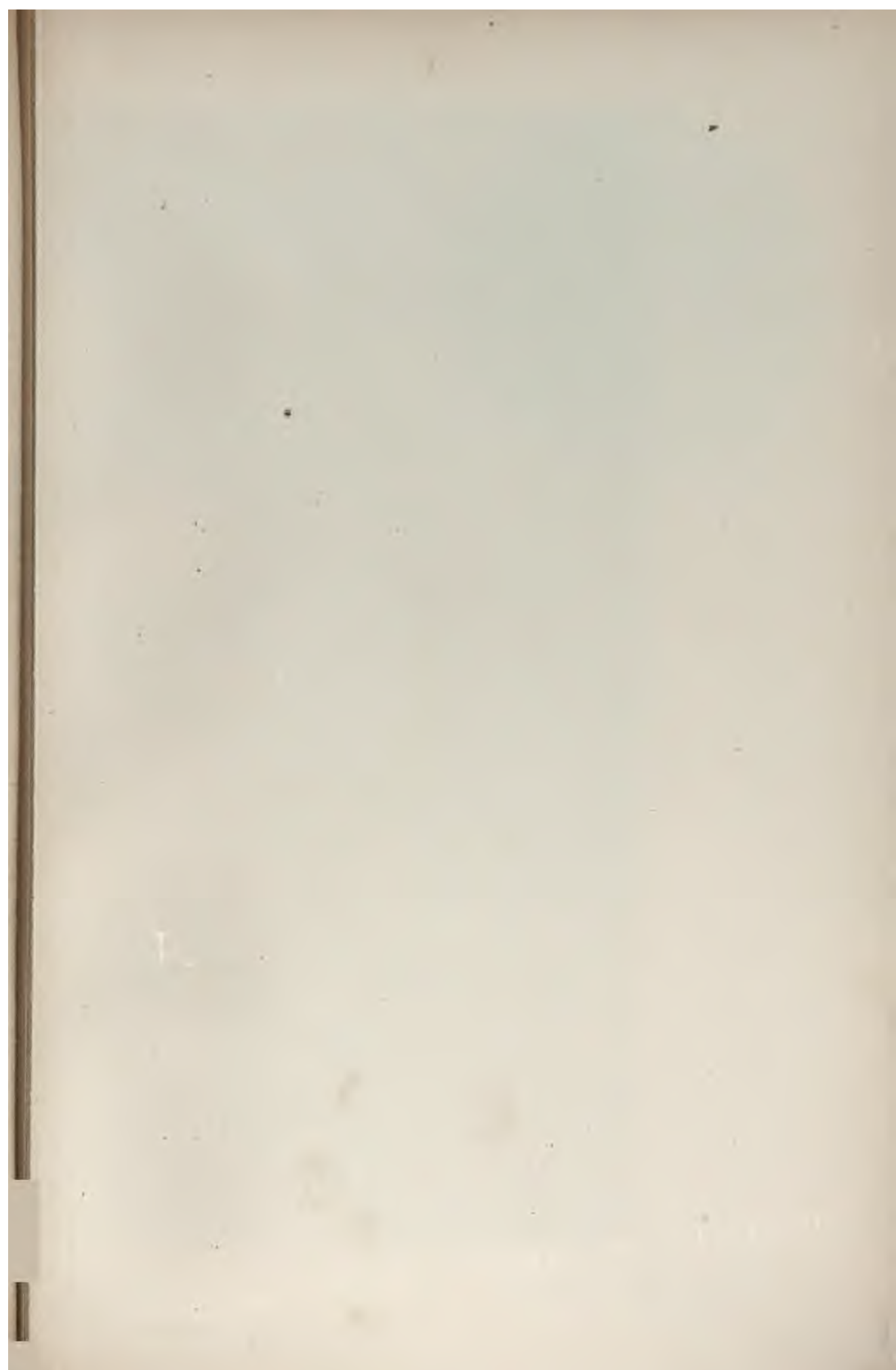
A. miliaris
magnified 3.



DeLafield

de
la

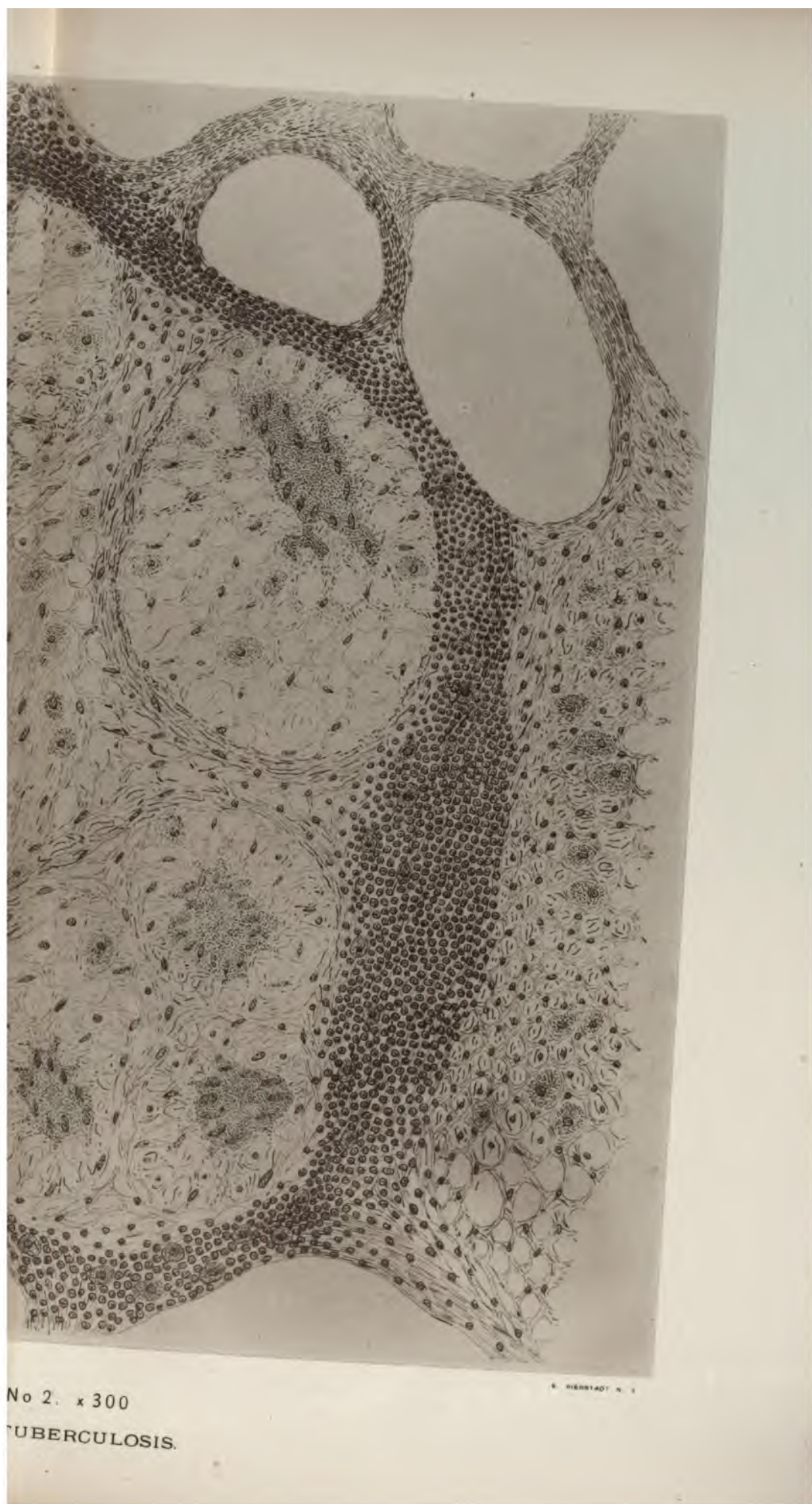






ARTOTYP

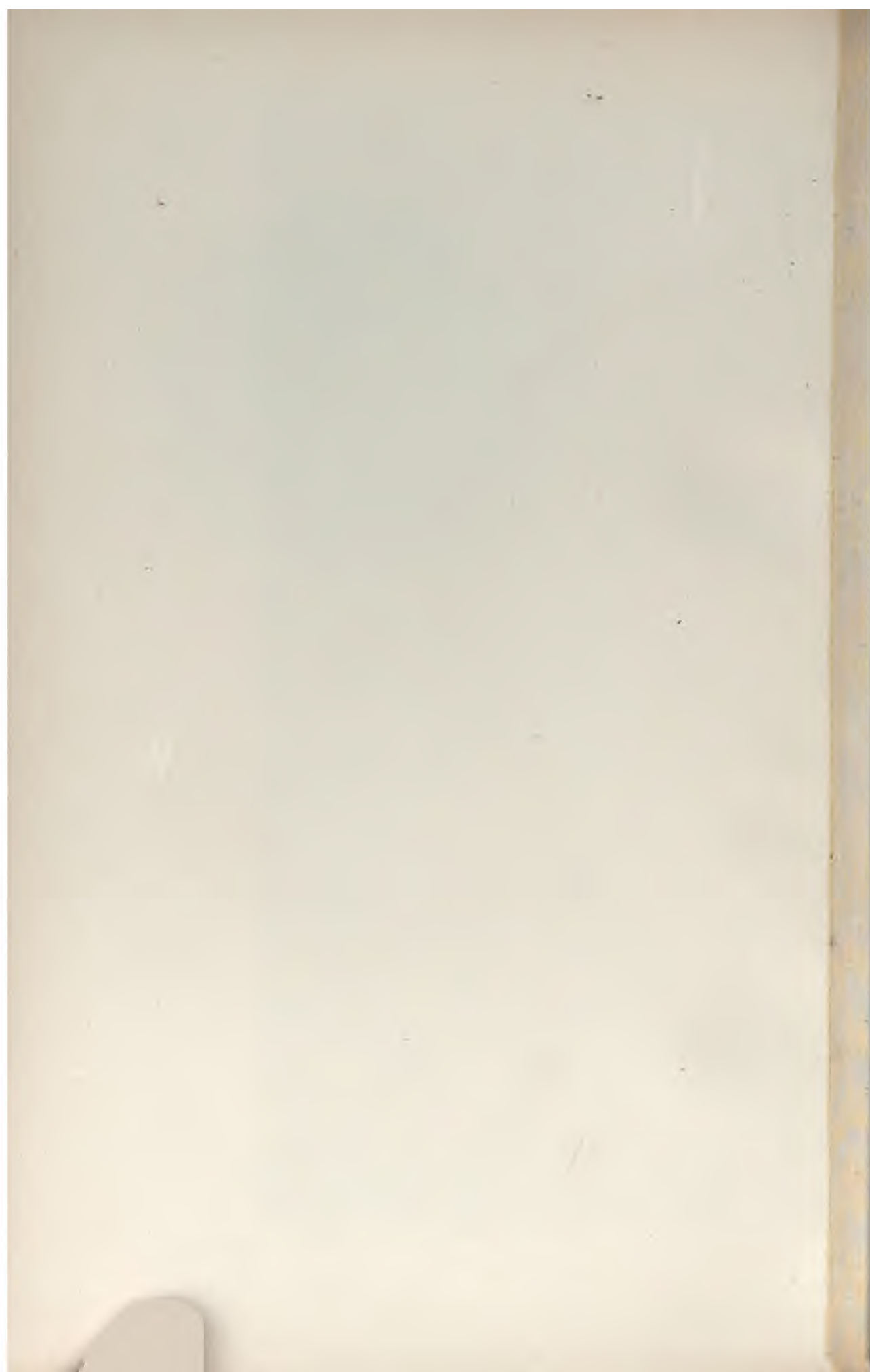
Plate XLIII
ACUTE MILIARY



No 2. x 300

TUBERCULOSIS.

S. HERBIADY N. 2



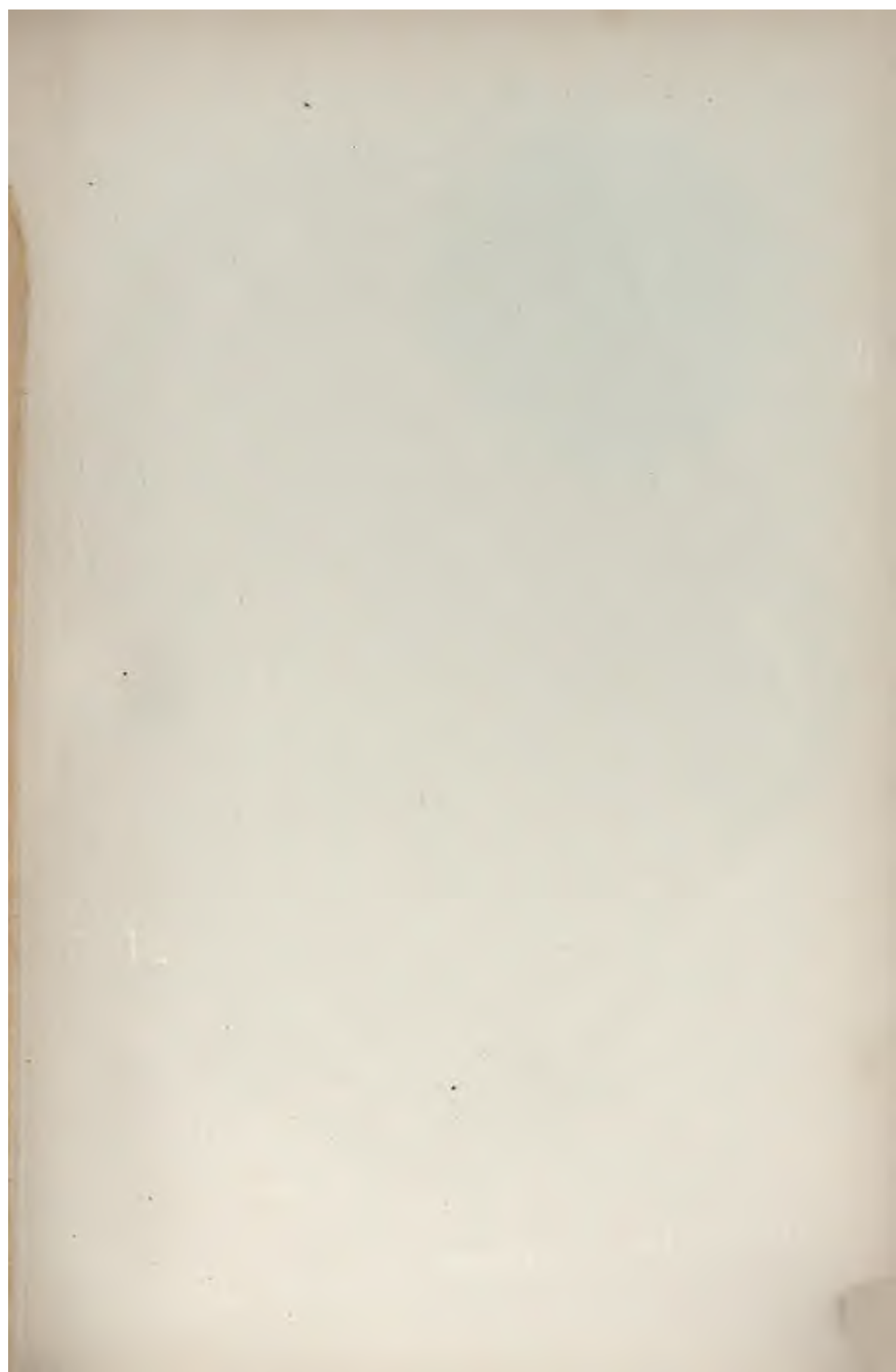




PLATE XLVII.

*An air vesicle forming part of a military
magnified 850 diameters.*

the lower and right hand portion of the plate are four large irregular spaces looking as if they were formed of the fusion of one or more air-vesicles and full of cells. On looking at these spaces more closely, it will be seen that one of them is filled with tissue identical with that composing the tubercle granula and diffuse tubercle; while the three others are only partly filled with such tissue, the rest of their cavities being filled with large epithelial cells, granular matter, and blood globules. In other parts of the field are air-vesicles filled with epithelial cells, and all the surrounding air-vesicles are lined with similar large epithelial cells. In these composite tubercles there are often air-cells of which the contents are somewhat puzzling. The walls of these air-vesicles are not thickened, their blood-vessels can be injected, the elements which fill the air-cell do not seem to be continuous with its wall. The cells contained within the air-vesicle looks more like those of tubercle tissue than like epithelial cells; they are imbedded in a finely granular basement substance. See Plate XLVII.

In this kind of miliary tubercles also cheesy degeneration may begin at the centre, and involve a large part of the tubercle.

It will be seen that these tubercles differ from the second variety, in that they are composed not merely of solid tissue replacing the air-vesicles, but that a large part of the tubercle is composed of filled air-vesicles. The tubercle tissue is essentially the same in both varieties—polygonal cells imbedded in a basement substance, and giant-cells, either present or absent. But in this third variety the production of epithelial cells, the filling of the air-vesicles with tubercle tissue, with epithelial cells, and with a tissue intermediate between these two, is much more marked. It is very difficult in either variety to see anything which looks like an early stage of infiltration of the wall of an air-vesicle.

These two varieties of tubercle do not usually occur in the same lung; but in different cases of acute tuberculosis the lungs are filled with one or the other variety, and it is hardly proper to look on them as different stages of the same process.

(4.) Miliary tubercles which consist simply of infiltrations of the walls of air-vesicles, of bronchi, of blood-vessels, and of lymphatics.

These tubercles are usually small and of irregular shape. Many of them are composed of diffuse tubercle, not of tubercle granula; they rarely contain giant-cells. Some of them seem to consist of nothing but small round cells imbedded in a basement substance.

These tubercles do not occur by themselves, but are found in larger or smaller numbers in the same lungs which contain the second and third varieties.

It has become the fashion to lay great stress on the absence of blood-vessels in miliary tubercles, and to ascribe the cheesy degeneration to the scantiness of the vascular supply. I think that there is some exaggeration in this. If we inject, artificially, lungs which are the seat of miliary tubercles, the injection does not indeed penetrate the tubercle granula, but it runs freely into the walls of the air-vesicles which form part of the miliary tubercle, and even into the diffuse tubercle. It is only the first variety of soft cheesy tubercles which cannot be thus injected.

If we look at the miliary tubercles which occur in other parts of the body, we find a repetition of what we have seen in the lungs.

(1.) Miliary tubercles, composed of nothing but amorphous granular matter, with pus-cells at their edges. Such tubercles are found especially in the pleura, peritoneum, liver, spleen, and kidneys.

(2.) Miliary tubercles, composed of one or more tubercle granula and of diffuse tubercle; around such tubercles are pus-cells and new connective-tissue cells in variable numbers. Such tubercles are found especially in the pleura, peritoneum, liver, spleen, bladder, lymphatic glands, medulla of bones, muscles, in syphilitic ulcers, in lupus, in granulation tissue, in the synovial membranes of the joints, in sarcomatous tumors.

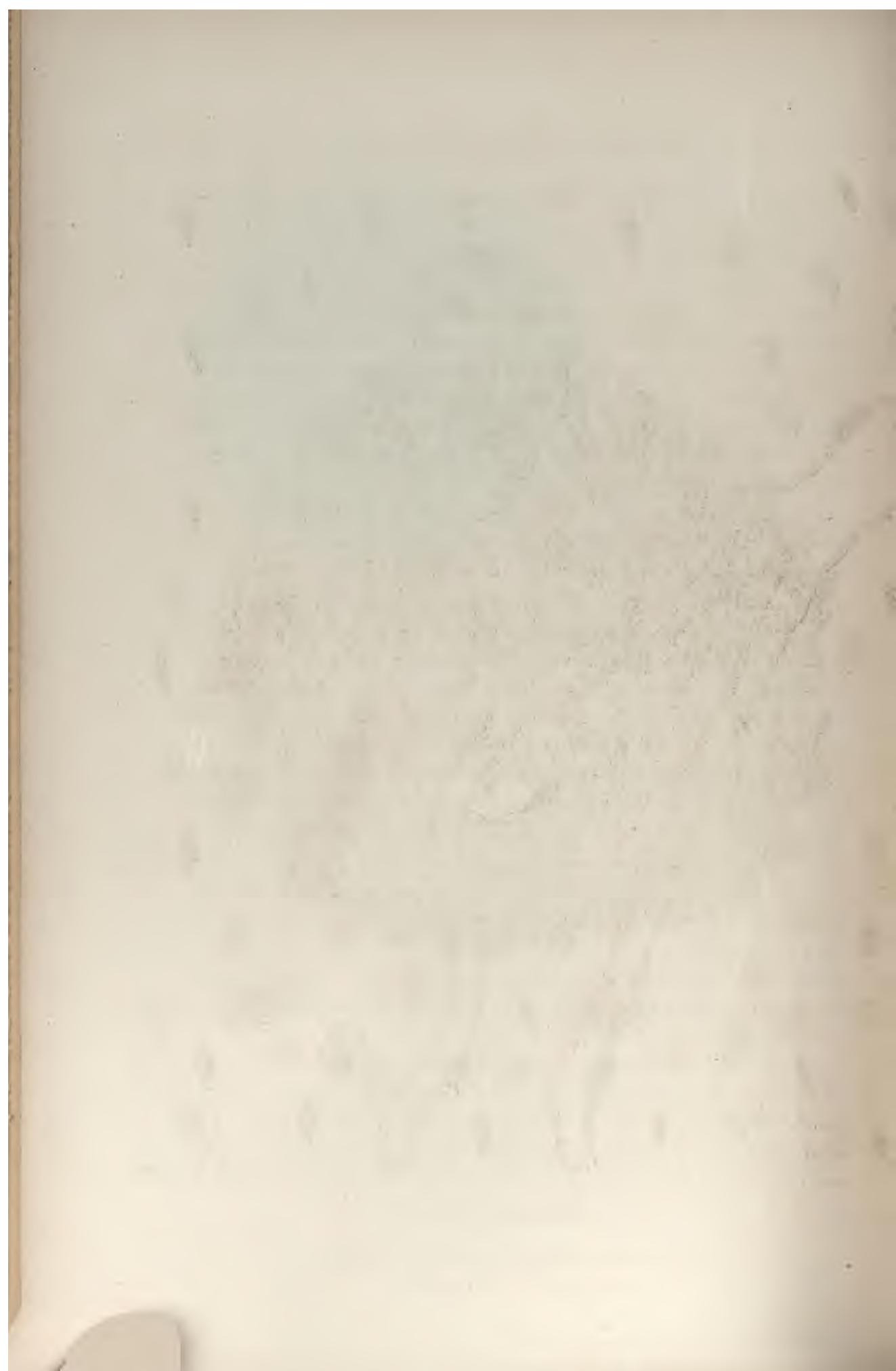
(3.) Miliary tubercles formed of an accumulation of large and small polygonal cells and of small round cells. These polygonal cells are much more distinct than the cells in tubercle granula; they seem to be held together by some kind of basement substance which I have never been able to demonstrate. These tubercles are found in the pia mater and omentum, and I have seen them once in the liver. They remind one of some of the air-vesicles in the third variety of the miliary tubercles of the lungs. See Plates XXVI. and L. In these tubercles there may be, large multinucleated cells, which look something, but not exactly,



De la field

PLATE I.

*A military Tubercle of the Bala Murci.
magnified 850 diameters.*



like the giant-cells found in tubercle granula. There is a well-marked growth of endothelial and connective-tissue cells in the pia mater and omentum in the neighborhood of such tubercles.

(4.) Miliary tubercles formed by an accumulation of small round cells around blood-vessels, between the perivascular sheath of the vessel and its wall. These tubercles are only found in the pia mater, unless some of the very small tubercles in the lungs are of the same nature.

(5.) Miliary tubercles apparently formed of nothing but an aggregation of small round cells, not to be distinguished from what is seen in ordinary inflammation, except by their being arranged so as to form little nodules.

We find, then, that the bodies, commonly known by the name of miliary tubercles, do not all have the same structure, so that this name does not designate any definite anatomical tissue, and in this sense it is proper to say that there is no regular structure belonging to tubercle. In the larger number of miliary tubercles, however, we do find a tissue of a particular kind which is found nowhere else, a tissue composed of polygonal and giant-cells imbedded in a basement substance. This tissue, as being the only anatomical characteristic of tubercle, we may call tubercle tissue; and according to the arrangement of its elements we may divide it into tubercle granula and diffuse tubercle. This, however, brings us to the somewhat curious conclusion that some tubercles are formed only of tubercle tissue, some of tubercle tissue and ordinary products of inflammation, some of cells resembling those of tubercle tissue, but not regularly arranged in a basement substance, while some contain no tubercle tissue at all. The temptation is great to deny the name of miliary tubercle to all the nodules which do not contain tubercle tissue, and it is to be hoped that further studies may enable us to do this, but for the present we have to abide by the conclusion stated above.

Thus far we have been concerned with the study of the structure of miliary tubercles; we must next inquire under what circumstances such tubercles are formed. We must distinguish, first, the cases in which miliary tubercles are found in large numbers scattered through different parts of the body—general tuberculosis; and the cases in which the

tubercles are confined to a particular part of the body—local tuberculosis.

The cases of general tuberculosis again must be divided into two sets, cases of chronic phthisis complicated by general tuberculosis, and cases of idiopathic general tuberculosis.

(1.) Idiopathic, acute, general, miliary tuberculosis. This disease occurs at all ages and in both sexes. Its duration varies between one and six weeks. It probably always terminates fatally. The temperature is usually between 99° and 103° , sometimes as high as 107° , occasionally never over 99° .

As a rule, cerebral symptoms—delirium, muscular twitchings, convulsions, and coma, exist during the course of the disease. The patients emaciate, their tongues become dry and brown, and they look like persons suffering from a continued fever. In addition to the general symptoms, there may be local symptoms corresponding to the organ in which the largest number of tubercles are developed. If there are many tubercles in the pia mater, there will be convulsive movements of the muscles of the face, general convulsions, difficulty in swallowing; the delirium and coma come on earlier. If the lungs are extensively involved, there may be pain in the chest, rapid breathing, cough, cyanosis, and subcrepitant râles. If the peritoneum is principally affected, there may be pain and tenderness over the abdomen and tympanites. In a large number of these cases, however, the symptoms are obscure and indefinite. The diagnosis must be made partly by exclusion, partly by familiarity with similar cases.

The opinion has prevailed that the cause of the disease in these cases is a focus of pus, in the condition of cheesy degeneration, in some part of the body. So many cases, however, occur in which no such focus exists, that such an opinion is no longer tenable. We certainly cannot account for all cases of general tuberculosis in this way, although it is quite possible that some cases may be thus produced.

(2.) Acute, general, miliary tuberculosis, occurring in persons already suffering from chronic pulmonary phthisis. In these cases the lesions and the symptoms are much the same as in the preceding set of cases, except so far as they are modified by the chronic phthisis. It

has been customary in these cases to regard the production of miliary tubercles as in some way secondary to the chronic phthisis, either from absorption of cheesy matters, or in some other way.

(3.) Acute, local, miliary tuberculosis occurring in persons already suffering from chronic phthisis. In these cases the miliary tubercles are usually confined to the lungs, the pleura, and the pia mater, but may also occur in other parts of the body.

(4.) Acute, local, miliary tuberculosis in persons not suffering from chronic phthisis. The miliary tubercles have been observed in the pia mater, the lungs, the costal pleura, the peritoneum, the bones, the synovial membranes of joints, in granulation tissue, in the walls and floors of old ulcers, of syphilitic ulcers, and of ulcers due to lupus. It is asserted that they have been seen in gummy tumors and in sarcomatous tumors of bone.

From the consideration of the anatomy of miliary tubercles, and of the circumstances under which they occur, it seems to me that we are driven to one of two conclusions.

(1.) We may believe that certain persons, either from the absorption of cheesy matter, from natural constitution, or from unknown causes, become tuberculous. They become the subjects of some general vital changes which produce symptoms and lesions, the lesions being miliary tubercles. The tubercles, however, have no essential anatomical characters, but vary in different cases. We do not say that a person is tubercular from the consideration of the minute anatomy of the lesions; but if we make up our minds, from the clinical history and the gross post-mortem appearances, that a person is tubercular, then we call all the little nodules that we find in that person miliary tubercles.

(2.) We may believe that there is a definite and essential anatomy belonging to miliary tubercles, which can be always recognized. Then we must admit that tubercle is composed of a tissue, like granulation tissue, on the border line between the new growths and the inflammatory products. This tissue is developed under a variety of circumstances, and is not confined to persons suffering from general tuberculosis, but occurs as a strictly local lesion. We must also admit that, in persons suffering from acute general tuberculosis, all the nodules found

after death are not composed of such tissue. In other words, tubercle is not a specific tissue necessarily implying the existence of a diathesis, but is a tissue of indifferent character, produced under a variety of circumstances. It may be the lesion of a general disease; or the lesion of a local, progressive inflammation; or an accidental lesion forming a non-essential part of local inflammations and new growths.

We may then continue to use the word miliary tubercle to designate all the little nodules which occur in tuberculous persons, irrespective of their structure, employing the terms tubercle granula and diffuse tubercle to the special tissue found in some of the nodules. Or we may hold that the nodules, although of different structure, are examples of the same tissue in different stages of development. Or we may deny the name of miliary tubercle to all the nodules which are not composed of characteristic tubercle tissue.

CHRONIC PULMONARY PHTHISIS.

THERE is hardly any disease which occurs as frequently as does pulmonary phthisis. Not only is it a common disease, but a fatal one. Year after year every hospital autopsy-room is filled with the bodies of persons who have died of phthisis. But despite of this large material for examination, not even the morphology of the lesions is thoroughly understood.

Just at the present time there are current three principal theories as to the nature of pulmonary phthisis.

First there is the older German school, which teaches that we can distinguish between an inflammatory phthisis and a tubercular phthisis; that, in many cases of phthisis, tubercle does not exist at all, or is developed secondarily to the inflammatory products; that most of the larger cheesy masses met with in the lung are ordinary inflammatory products. This theory has led to the use of such names as catarrhal, caseous, and interstitial pneumonia; peribronchitis nodosa; catarrhal and fibrous phthisis, to designate the different lesions.

Secondly there is the modern French school, which teaches that the essential and primary lesion of all phthisis is tubercle; that the accompanying inflammatory lesions are comparatively unimportant; that an inflammatory phthisis without tubercle does not exist. Some even go so far as to deny that simple inflammatory products ever undergo cheesy degeneration.

Lastly, there is the younger German school, which denies that there is any characteristic lesion at all belonging to phthisis, and holds that the only test is their inoculability. Whatever lesions can be inoculated in animals and produce in them miliary tubercles, are to be regarded as tubercle, no matter what their anatomy may be. Added to this theory is the belief that a specific form of bacteria is the exciting cause of the productions of tubercles.

Besides these principal theories there are innumerable modifications of them.

My particular object is not to advocate any one of these theories, nor to advance a new one. It is simply to describe and figure what I have been able to see in the lungs of persons who have died with chronic phthisis.

When we look at the lungs of such persons, we observe a number of lesions. Some of these lesions look as if they were not related to each other; others look as if they represented different stages of the same process. The clinical histories of these patients also enable us to judge that some of the lesions are recent and that others have existed for a long time.

In the ordinary forms of the disease we find :

1. Lungs studded with small nodules like the nodules which we see in acute miliary tuberculosis, but the nodules are larger, harder, and look older.

2. Lungs studded with similar nodules, but the nodules are joined together or aggregated into masses by a dense tissue. At the same time more or less of the lung is converted into a dense, fibrous-looking mass.

3. Lungs studded with white or yellow lobules, surrounded by red or gray hepatization.

4. Lungs in which are large, dense, cheesy masses.

5. Lungs which look as if they consisted entirely of fibrous tissue and cheesy nodules.

6. Lungs in which these different lesions are associated.

Besides we find cavities of different sizes and in different conditions associated with these different lesions.

The cases characterized by the presence of small nodules, either with or without diffuse fibrous-looking tissue, form a group by themselves.

The cases characterized by the presence of white or yellow lobules, form another different group.

The first group we will, for convenience, describe under the name of "Chronic Miliary Tuberculosis."

CHRONIC MILIARY TUBERCULOSIS.

This form of chronic phthisis has hardly received the attention which it deserves. At the present time, while the strife is going on between the partisans of an inflammatory phthisis on the one hand, and those of a tubercular phthisis on the other, study has been principally directed to the lobular infiltrations which we shall have to describe later. Chronic miliary tuberculosis has been rather summarily classed as a sequence of pneumonic phthisis; as representing tubercles which have become obsolete; or as a form of fibrous phthisis. And yet, in New York, at least, it is a very common form of chronic phthisis, and is seen in patients of many different nationalities.

The cases, however, are not all alike, and it is convenient to distinguish,

1. Miliary tubercles scattered singly through the lungs.
2. Miliary tubercles scattered through the lungs, but joined together by diffuse fibrous-looking tissue, or aggregated into masses by the same tissue, and often accompanied by the presence of large masses of the same fibrous-looking tissue.

I. Miliary tubercles scattered singly through the lungs.

The patients who suffer from this form of phthisis exhibit symptoms very much like those of patients who are the subjects of emphysema and chronic bronchitis. There is the same dyspnoea; the same cough with mucous, or muco-purulent expectoration; the same gradual emaciation; the same physical signs. But the loss of general health is more marked, and hæmoptyses, hectic fever, and night-sweats are

added to the other symptoms. The lesion seems to be a progressive one, but the progress is not always continuous. The history is often one of repeated attacks separated by considerable intervals, and after death the lesions in the lungs look as if they were not of the same date, but had been developed at different periods. Sometimes a patient will die of apoplexy or some other disease, while the tubercles are in an early stage of formation.

These cases are often very obscure and difficult of diagnosis. The absence of any physical signs except those of emphysema, bronchitis, and pleurisy, adds to the difficulty. Most of the patients die, not from the tuberculosis, but from some intercurrent disease.

If we can credit the testimony of some patients, the disease may exist without producing any symptoms.

After death we find old adhesions between the pulmonary and costal pleura; the lungs large, emphysematous and pale, unless pneumonia, congestion, or œdema exist as complications. The bronchi contain muco-pus; their mucous membrane is often congested and trabeculated; their entire walls may be thickened.

The whole or parts of the lungs are studded with small, white or grayish, hard, fibrous, or cheesy nodules, from the size of a pin's head to that of half a pea. These nodules are single, not joined together by diffuse tissue, not aggregated in patches. At the apices of the lungs, however, there may be older tubercles and a little diffuse fibrous tissue. Sometimes the tubercles are unusually close together, and there may then be a little diffuse tissue joining them together in a sort of network.

Not infrequently there is a growth of fibrillated connective tissue around the bronchi and blood-vessels, in the walls of the air-vesicles, and in the interlobular septa. On section these growths may give irregular figures somewhat resembling those of the miliary tubercles.

There may be also miliary tubercles in the pleura, the peritoneum, the liver, the spleen, and the kidneys.

It is somewhat curious that, even in the cases in which the tuberculosis is general, there are no such marked and fatal symptoms as exist in acute miliary tuberculosis.

II. Miliary tubercles accompanied by the growth of a diffuse fibrous-looking tissue.

This form of phthisis is very common. It is often spoken of as "fibrous phthisis." It must be remembered, however, that dense tissue, looking like fibrous tissue, is produced in the lungs in large amount simply as the result of an interstitial pneumonia, without the production of tubercle. In old lobular pneumonic phthisis, also, where cavities have been formed, the tissue between the cavities may look like fibrous tissue. It is usually not possible to determine by the naked eye the real nature of the fibrous-looking tissue which we see in such lungs. Whether it is fibrillated connective tissue alone, whether it is tubercle-tissue, or whether air-vesicles can still be seen in it, is only to be made out with the microscope.

The cases of this kind of phthisis run a slow course. The disease may be arrested after involving a small part of the lung, or it may go on until the larger part of both lungs is rendered unfit for use. When only a small part of the lung is involved there may be no rational symptoms (if we can believe the patients) and no physical signs.

The disease may begin insidiously, or it may be ushered in by one or more attacks of hæmoptysis. For a considerable time the symptoms are apt to be very obscure. The patient's general health suffers, but there may be no cough, no febrile movement, very little dyspnoea, no physical signs.

When, however, more of the lungs is involved by the lesions, the patients lose flesh and strength; cough, dyspnoea, and a febrile movement are developed; the pleuritic adhesions, the bronchitis, and the consolidation of the lungs give their appropriate physical signs.

The pleurisy, the laryngitis, the bronchitis, or the ulceration of the small intestines, which often exist as complications, may give rise to symptoms more prominent than those which belong to the lung lesion.

The fatal cases finally die in a condition of extreme emaciation.

The whole course of the disease is such as to give the impression of its being a chronic local inflammation of the lungs. The entire recovery of some patients and the regular proportion between the development

of the lesions and the intensity of the symptoms favor such a view. On the other hand, the formation of similar lesions in other parts of the body seems to stamp it with the character of a general disease.

After death we find the characteristic lesions developed to a greater or less extent.

Organized pleuritic adhesions are the rule, and with these there may be thickening of the pulmonary and costal pleura. Miliary tubercles also are not infrequently found in the pleura.

The bronchi show the changes common to chronic bronchitis, or there may be tubercle-tissue formed in their walls, or there may be ulcers. Less frequently they are the seat of croupous inflammation.

If the disease is not of long standing, we find at the apex of one of the lungs a few small, hard nodules joined together by a little solid tissue, or imbedded in a larger mass of fibrous-looking tissues. The adjacent air-vesicles are irregularly dilated, and bands of fibrous tissue run off from the central mass.

If the disease has existed for a longer time, the nodules are present in greater numbers, are distributed over a larger portion of the lungs, and look as if they had been developed at different periods. Some are of about the size of a pin's head, hard, grayish; others are larger, harder, and look more like fibrous tissue; others are large and cheesy, and may be softened at their centres. These nodules are joined together by solid tissue so as to form a sort of network, or they are imbedded in masses of solid tissue. This solid tissue is sometimes pigmented and the masses are then often called "patches of slate-colored induration."

In other cases there is developed a diffuse growth of fibrous-looking tissue, more or less pigmented, which replaces a considerable part of the parenchyma of the lung.

In the old cases a large part of the lung-tissue may be thus replaced by the miliary tubercles and diffuse solid tissue. Not infrequently, cavities of large size are also formed, especially at the apices of the lungs.

There are also changes in the lung-tissue between the tubercles. General emphysema may exist, or irregular dilatation of the air-vesicles



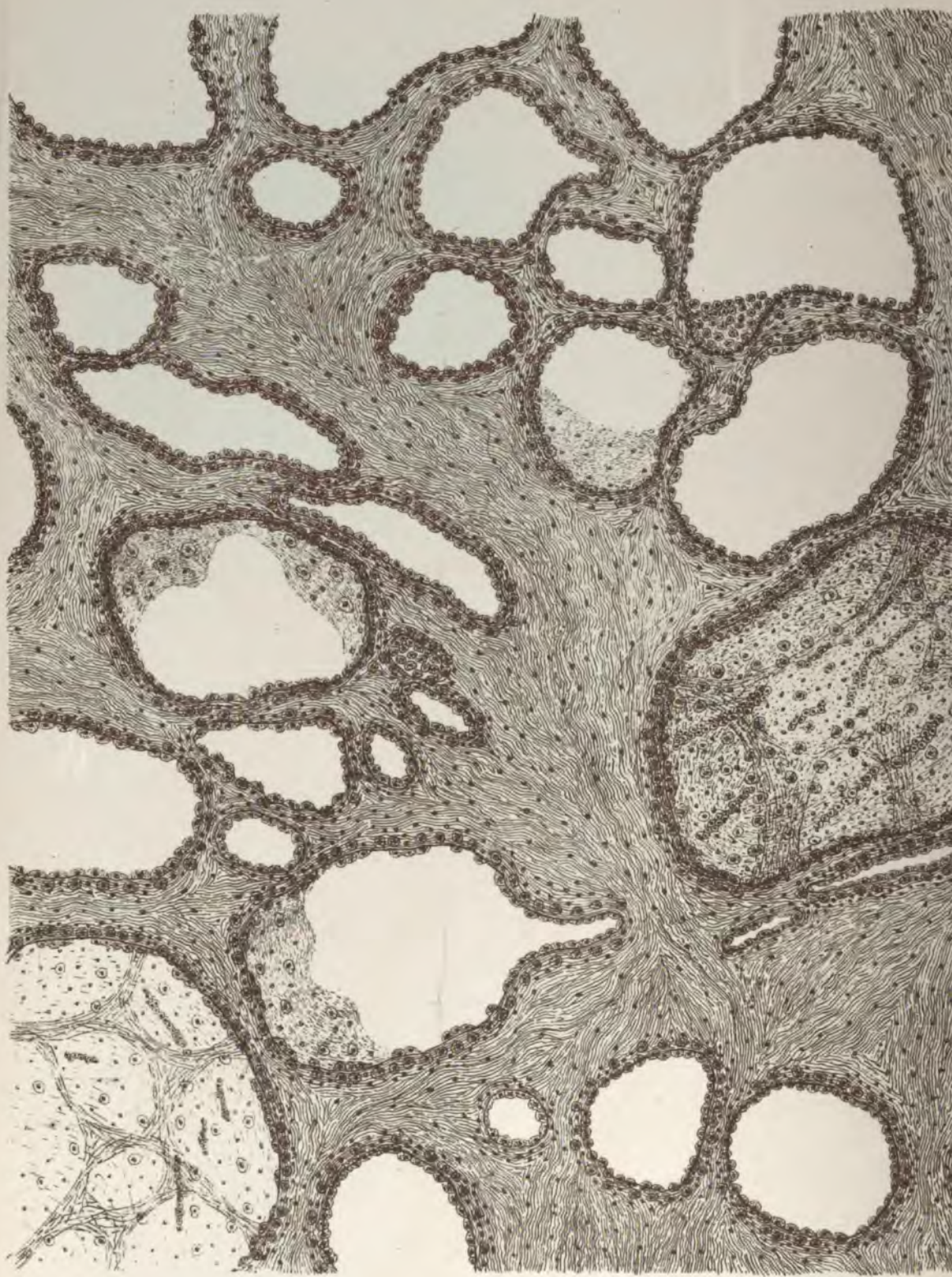
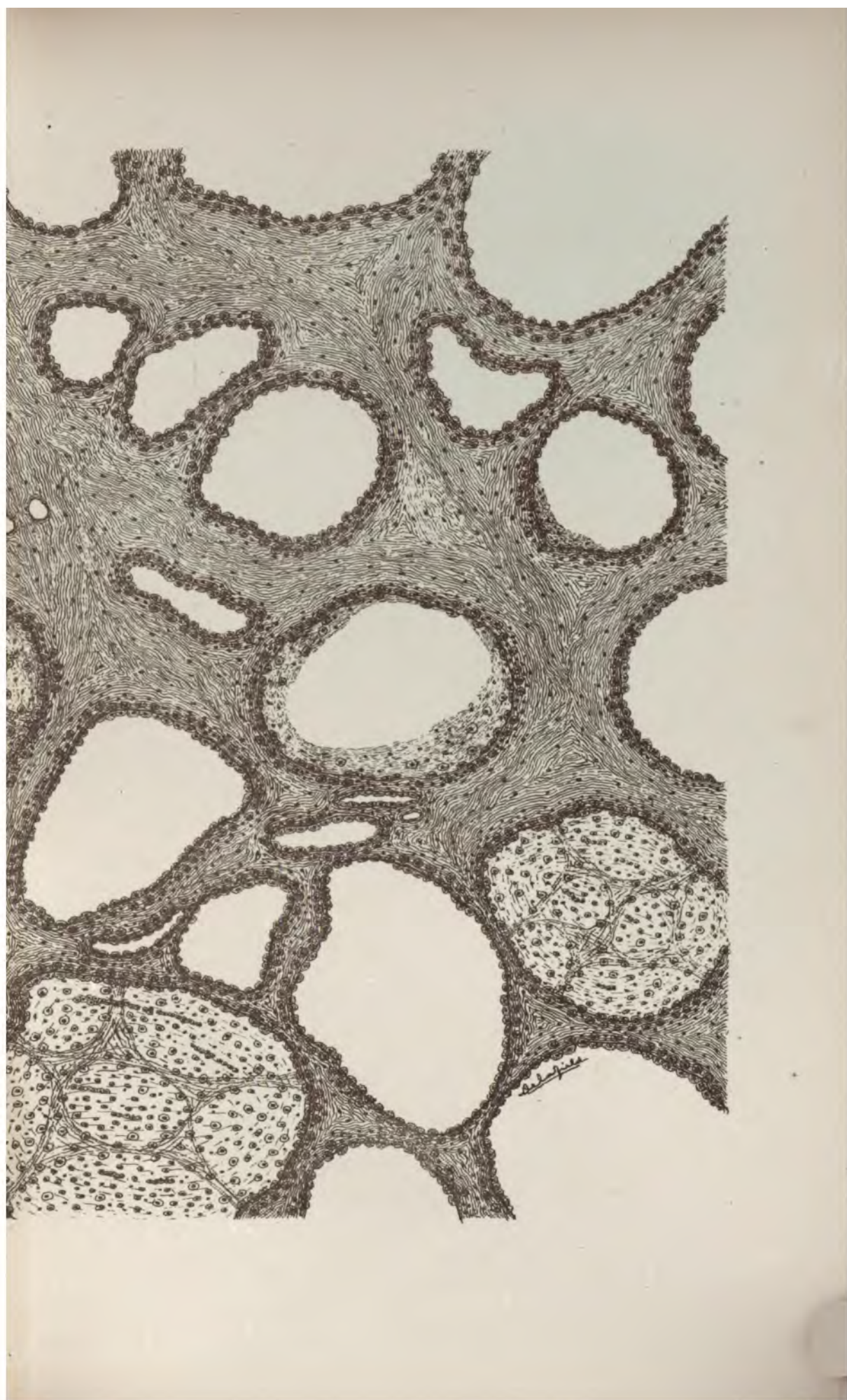


PLATE LXIII.

*Interstitial Pneumonia
magnified 90 diameters*





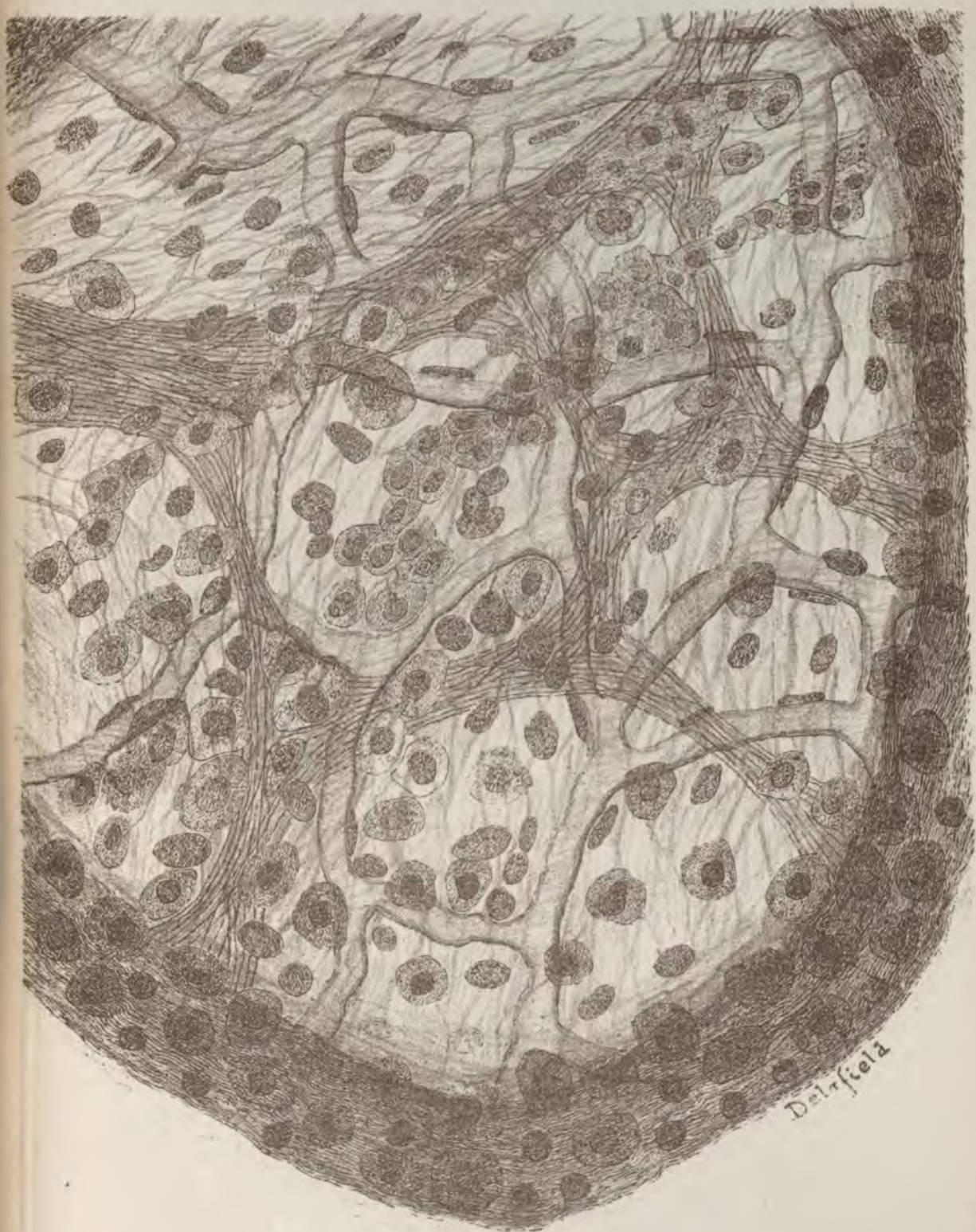


PLATE LIII.

*Cell Growth on the wall of an Air Vesicle.
magnified 850 diameters.*



adjoining the tubercles. Interstitial pneumonia, with a production of fibrillated connective tissue in the walls of the air-vesicles, the bronchi and the blood-vessels, is a frequent complication. There may be a growth of polygonal nucleated cells on the walls of the air-vesicles, and the cells may be numerous enough even to fill the vesicles..

The patients may be attacked by ordinary lobar pneumonia, as an intercurrent disease.

Tubercular lesions are often present in other parts of the body, especially in the larynx and trachea, the small intestine, and the peritoneum.

The anatomy of the miliary tubercles is the same in the two sets of cases of which we have been speaking. The difference between the two sets of cases consists in the presence or absence of the diffuse, fibrous-looking tissue.

It is convenient to describe separately the diffuse tissue and the miliary tubercles.

Of the nodules which have the gross appearance of miliary tubercles we can distinguish :

1. Nodules formed of fibrous tissue.
2. Nodules formed of tubercle-tissue.
3. Nodules formed of tubercle-tissue in different stages of degeneration.

(1). The nodules formed of fibrous tissue are composed of a dense, fibrillated basement substance in which are imbedded a few cells. The nodules are of irregular shape and are situated around the bronchi and blood-vessels, or in the walls of the air-vesicles. These nodules seem to be only part of a process of interstitial pneumonia which is in some lungs associated with the production of the true tubercles. Such an interstitial pneumonia not only forms the nodules spoken of, but also such a diffuse thickening of the walls of the vesicles as is seen in Plate LXIII. There is at the same time a change in the size of the air-vesicles: some are dilated, others are small and deformed. New cells are formed on the walls of the vesicles. Some of these are the large, polygonal, nucleated cells which we see in many forms of pneumonia. Others are smaller, are arranged in rows, and look like the new cells in inflamed

connective tissue. Plate LVIII. represents part of the wall of such an air-vesicle. The blood-vessels have been artificially injected. All the cells seen in the drawing were adherent to the wall of the vesicle, not free in its cavity.

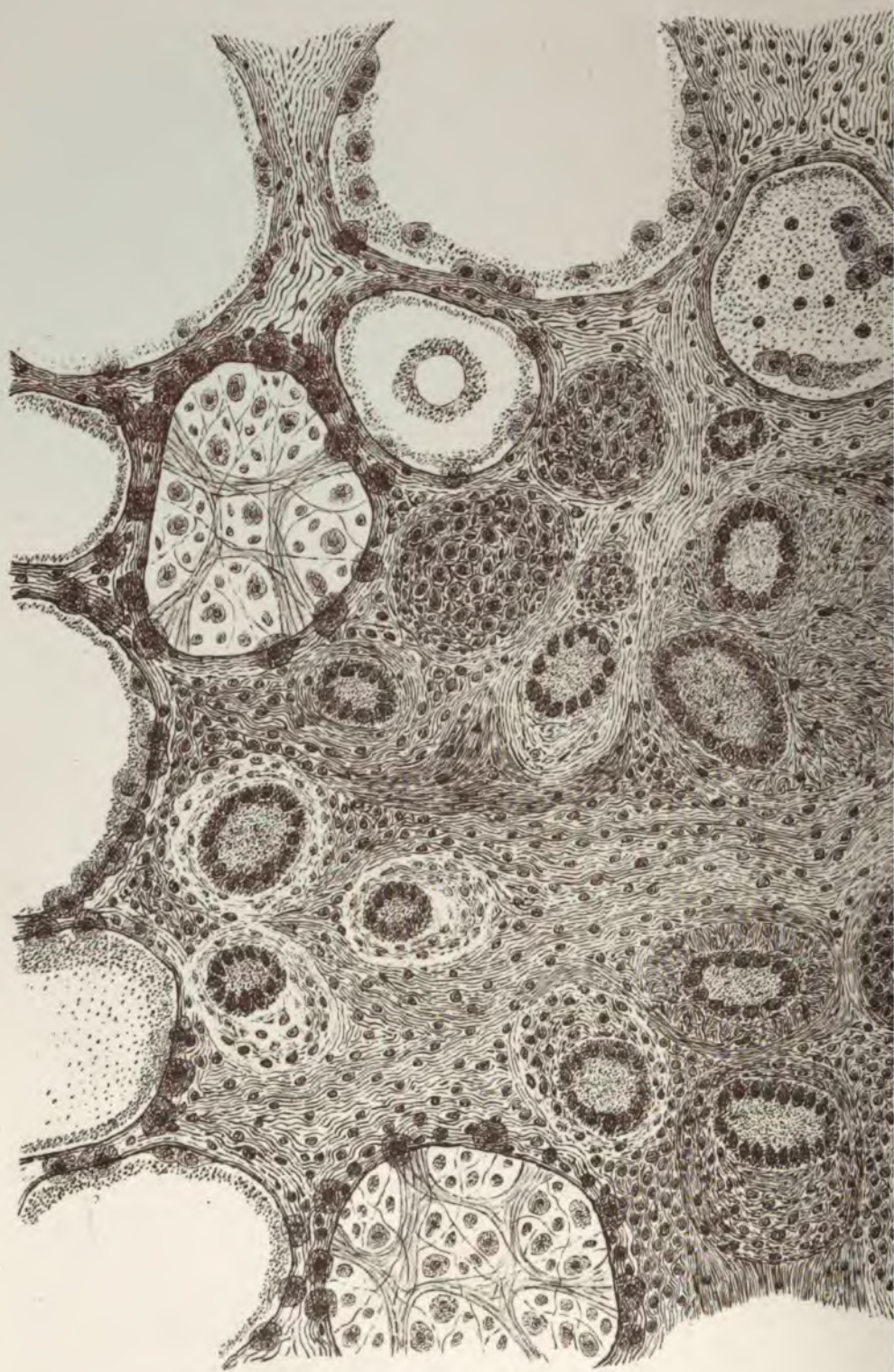
(2). Nodules formed of tubercle-tissue. These nodules are of two varieties:

(a). The simplest nodules, which have such a structure as is represented in Plate LI. Such a nodule, to the naked eye, is of a globular shape, but when magnified is seen to be of the irregular form represented in the plate. It will be seen that the nodule is composed of tubercle-granula surrounded by a dense tissue—partly tubercle-tissue, partly connective tissue. The shape and arrangement of the granula is such as to make it probable that they have been formed within the air-vesicles. This method of formation is much plainer in chronic than in acute miliary tuberculosis. The granula have the same structure as those found in acute miliary tuberculosis, but their basement substance is denser and more abundant, the cells are less numerous and often smaller. The basement substance may be much in excess and homogeneous, as seen in Plate LXII., or it may be fibrillated. The cells preserve their regular polygonal shape, or appear as round and oval nuclei. The giant cells may be present or absent.

The diffuse tissue between the granula also has the same structure as in acute tuberculosis, but here again the basement substance is more abundant and in places is replaced by ordinary connective tissue.

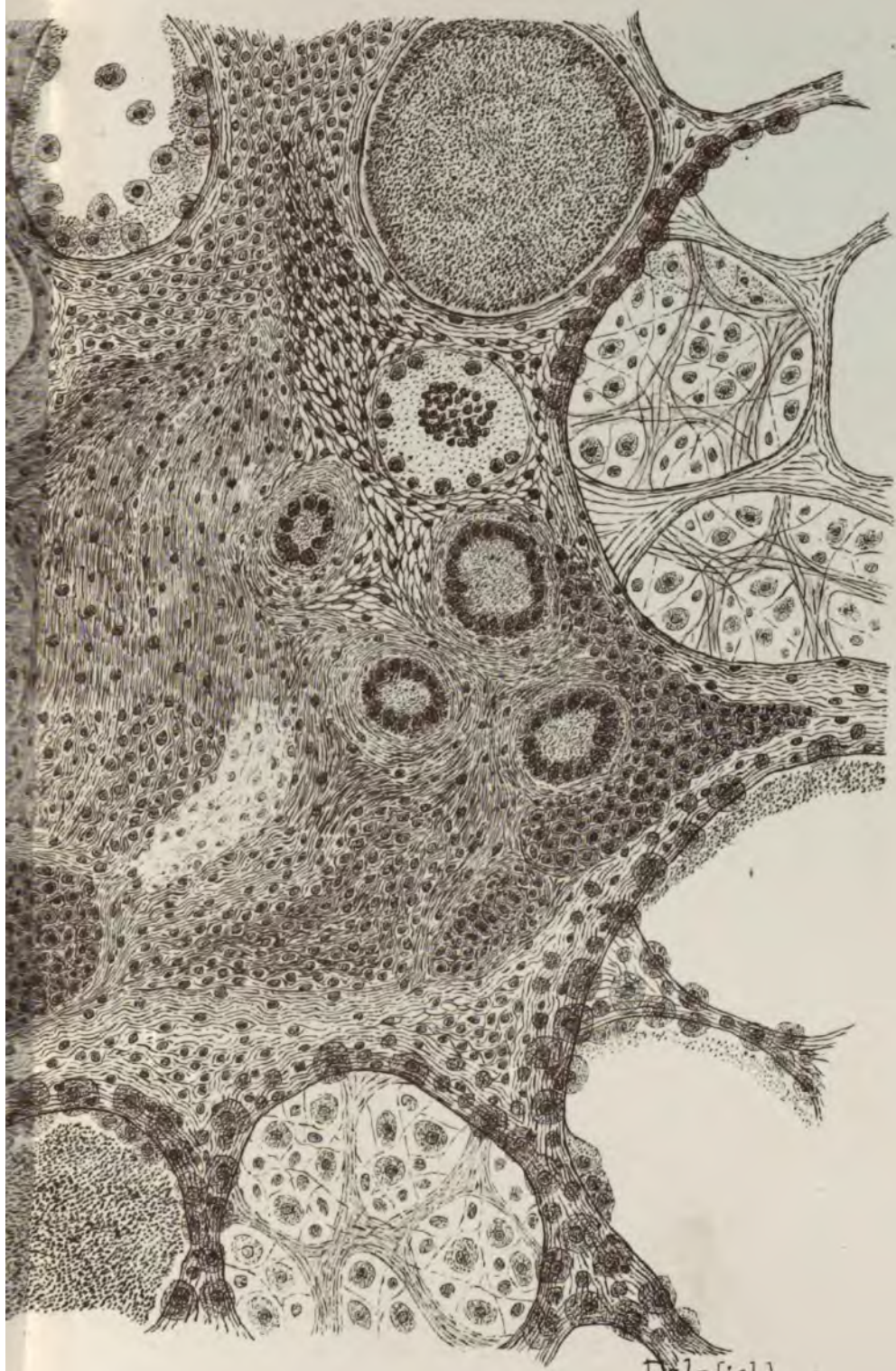
(b.) Nodules of more complex structure. The central portions of such nodules have the same structure of tubercle-granula and diffuse tubercle as those just described, but in addition to this they are surrounded by a zone of new tissue. Plate LVIII. represents a single tubercle of this kind, of which the blood-vessels have been artificially injected. The central portion of the tubercle is composed of tubercle-granula and tubercle-tissue, but there is a zone of small-celled tissue surrounding this. Plate LVI. represents a group of tubercles of which the peripheries are formed of the same small-celled tissue, and this same tissue joins some of the tubercles together.

This zone of new tissue forming the periphery of this variety of



PLA

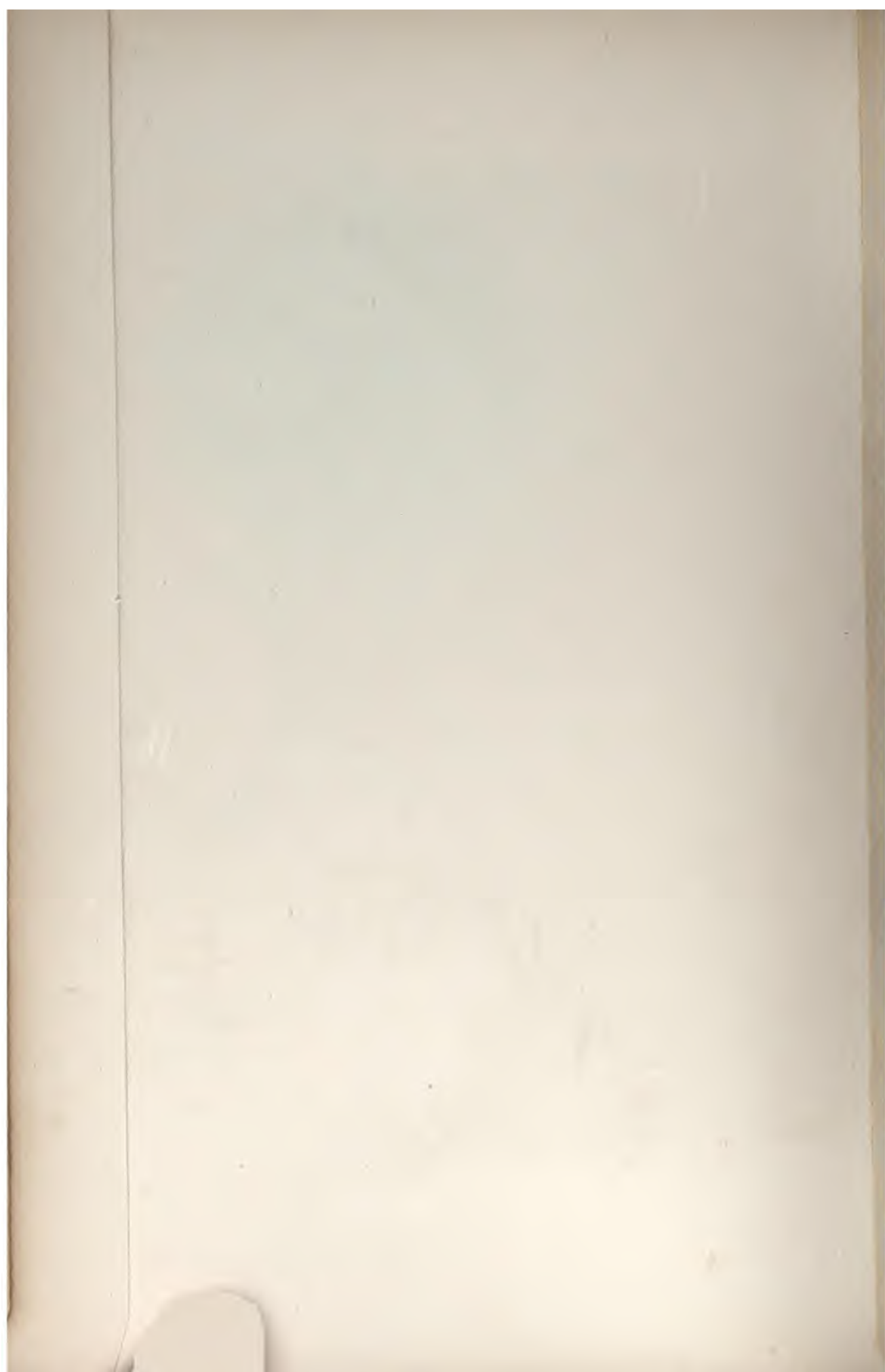
*A single m
magnifica*



De lafield

LI.

Tubercle
carcinoma



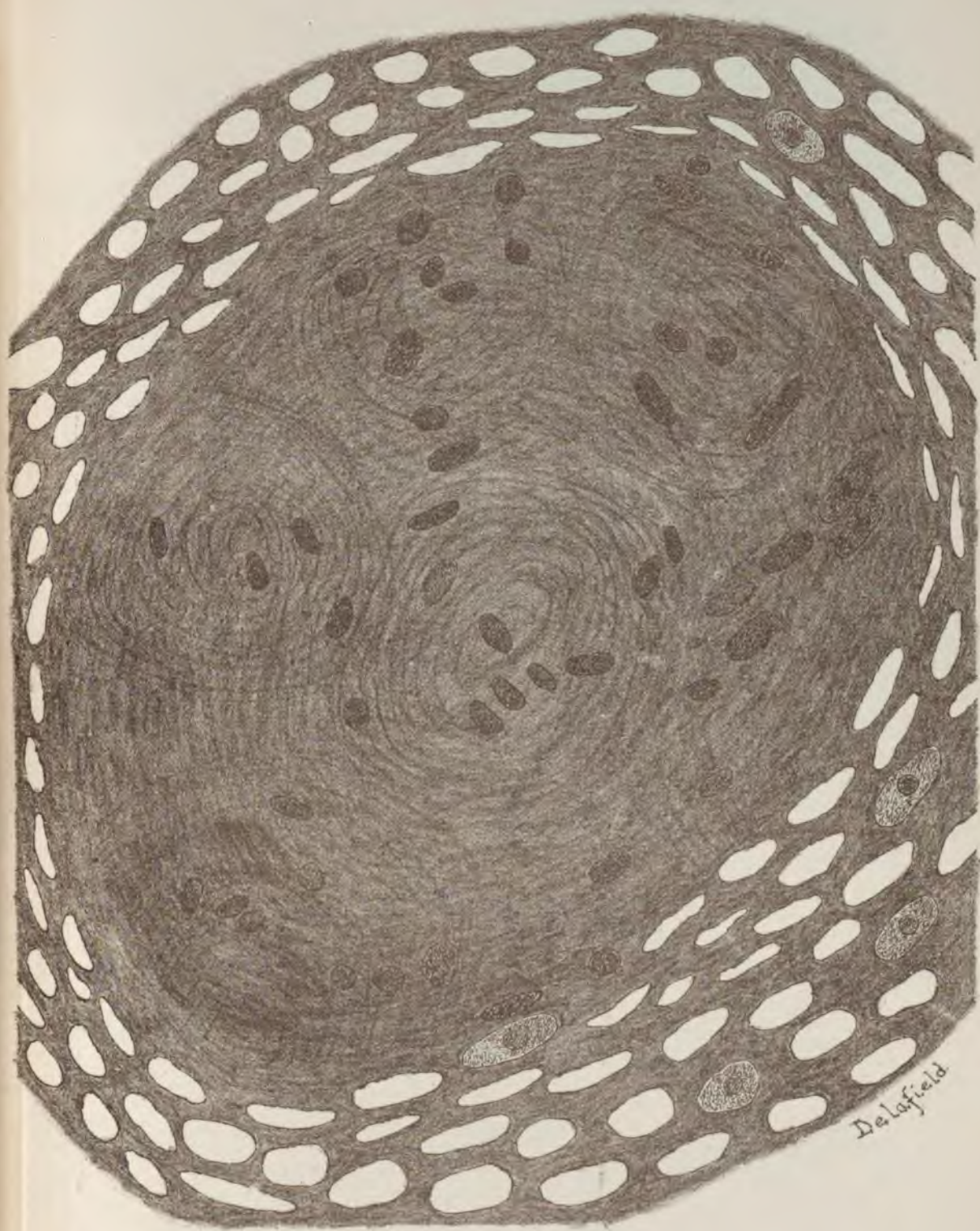
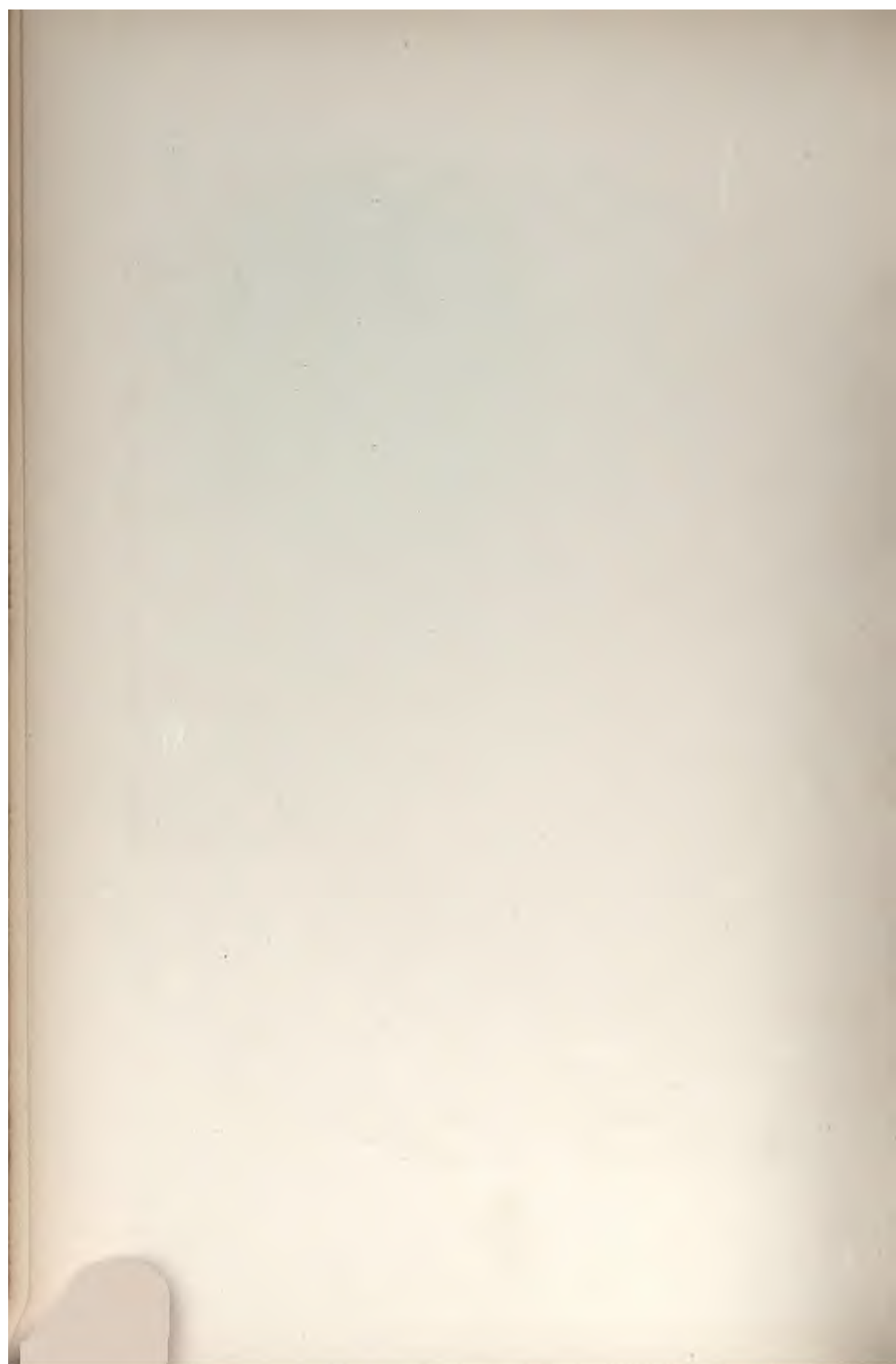
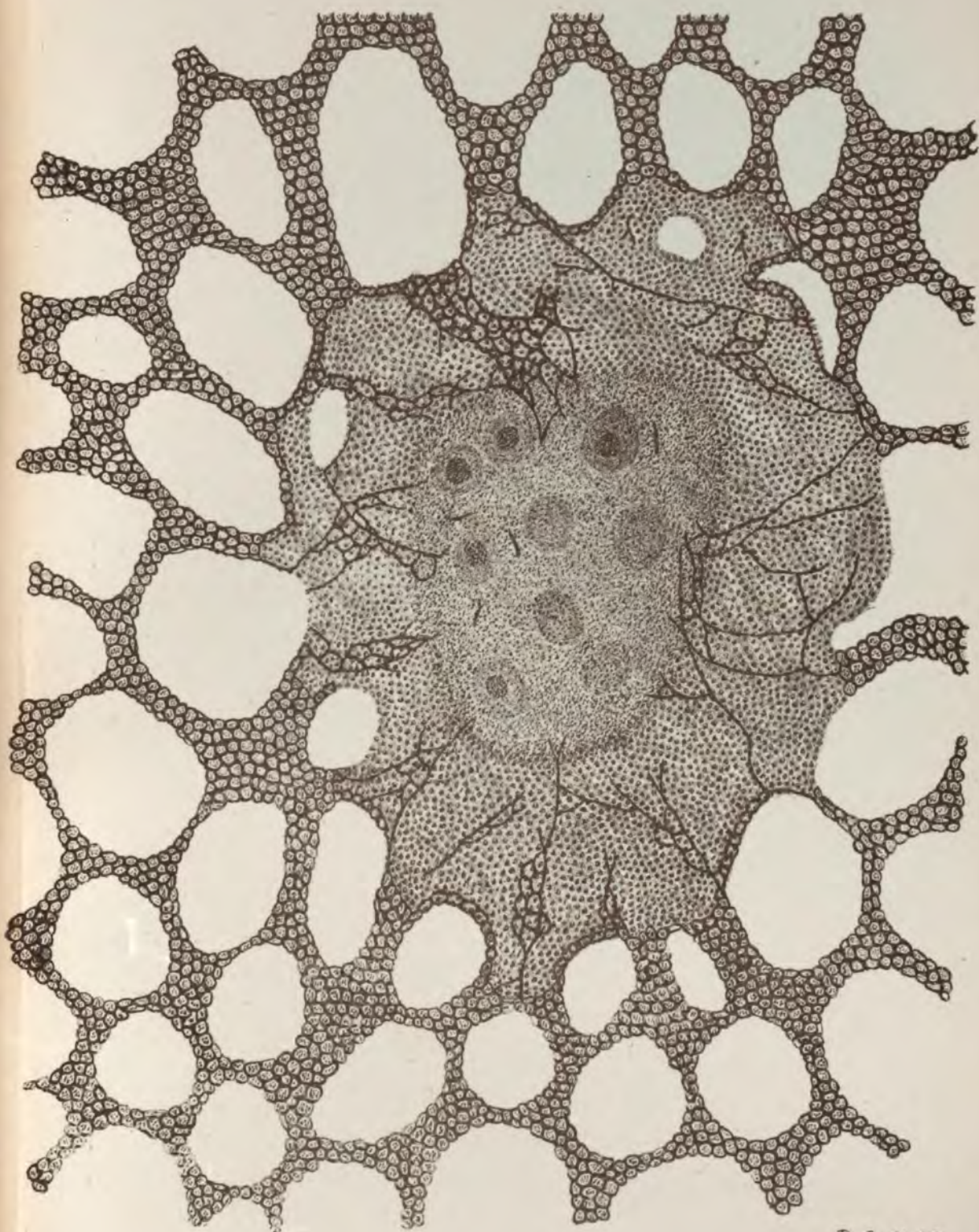


PLATE LXII.

*A Tubercle Granulum;
magnified 850 diameters*

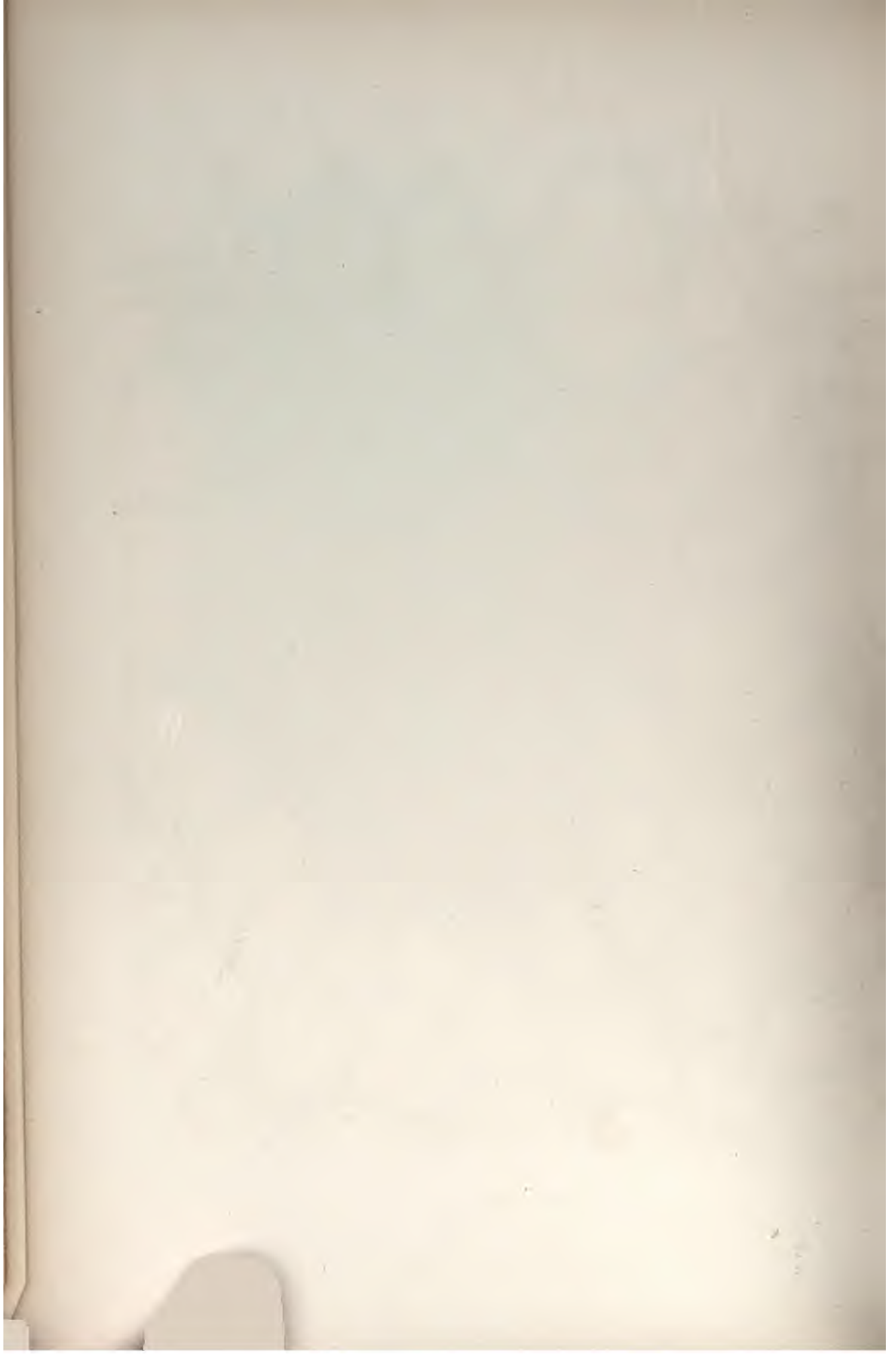




Delafield

PLATE LVIII.

*A military Tubercle;
magnified 90 diameters.*



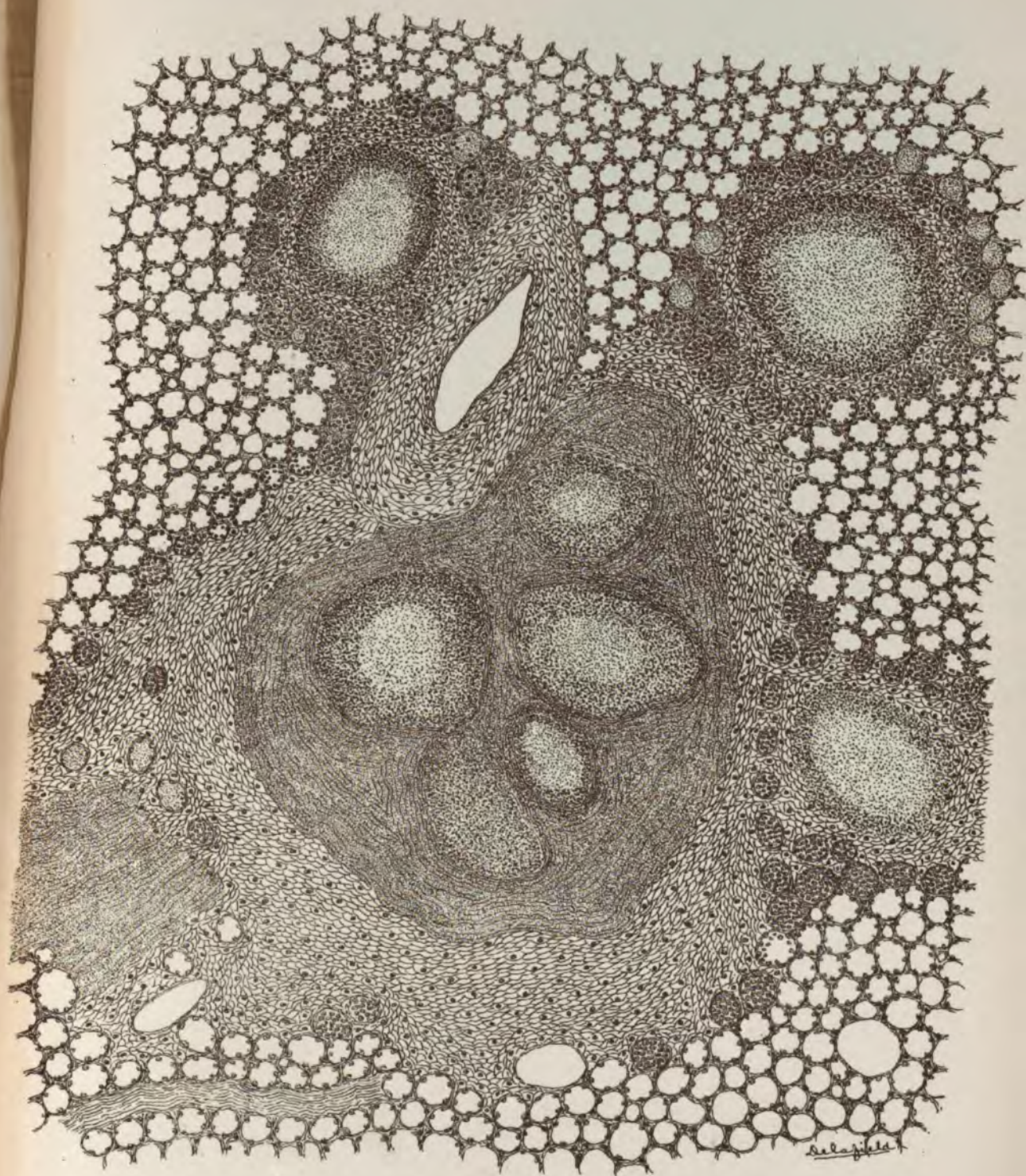
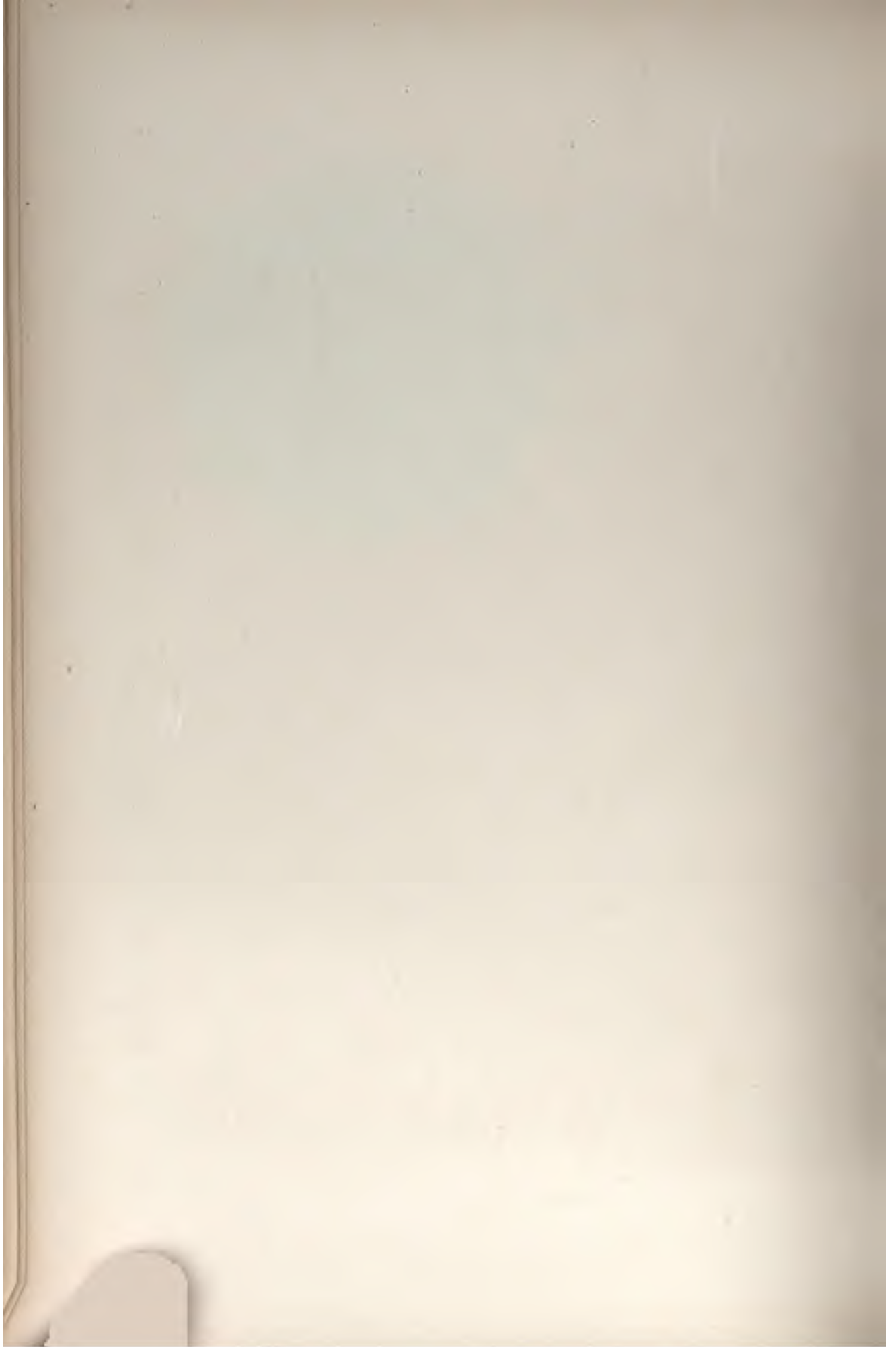


PLATE LVI.

*Miliary Tubercles;
magnified 90 diameters.*



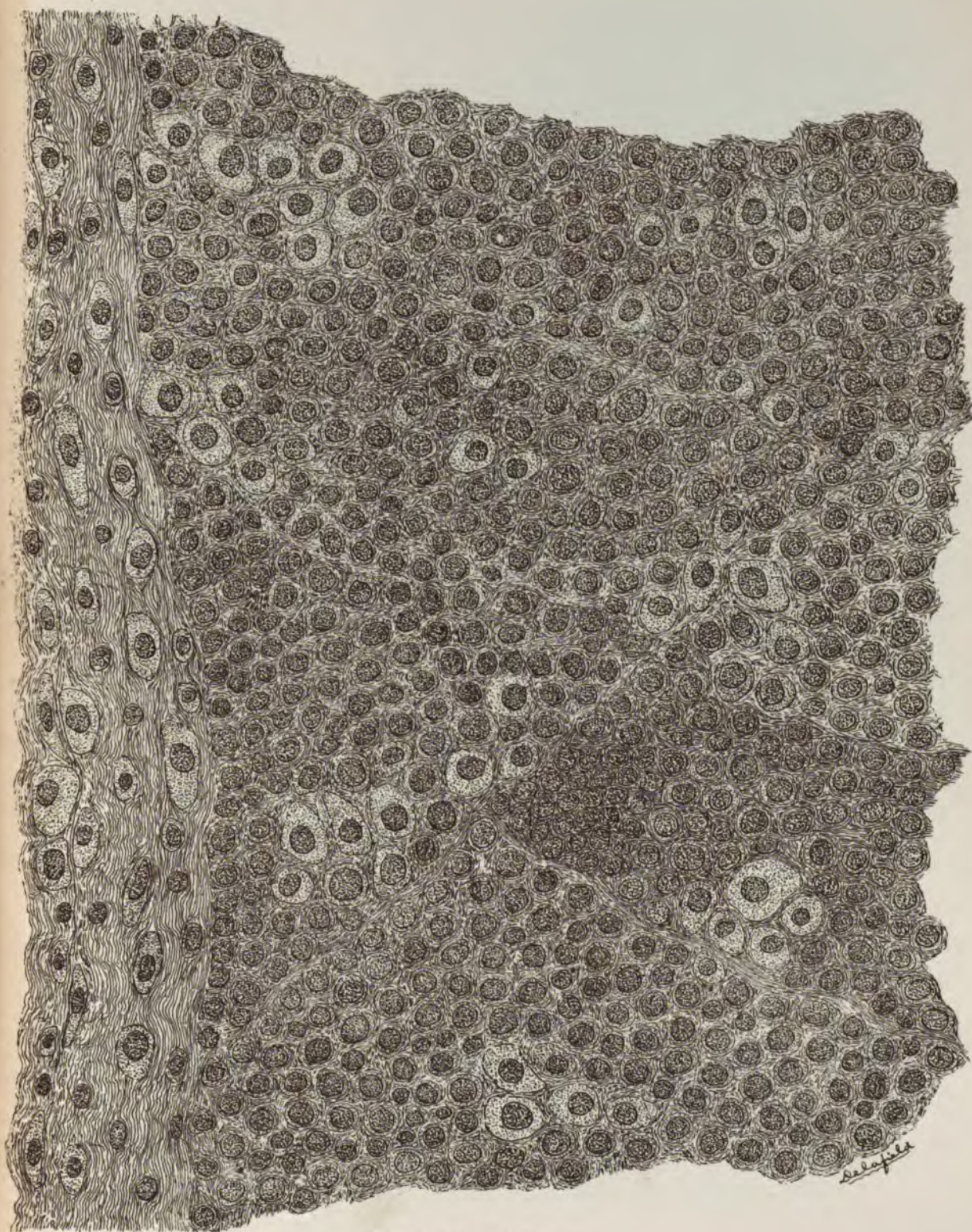
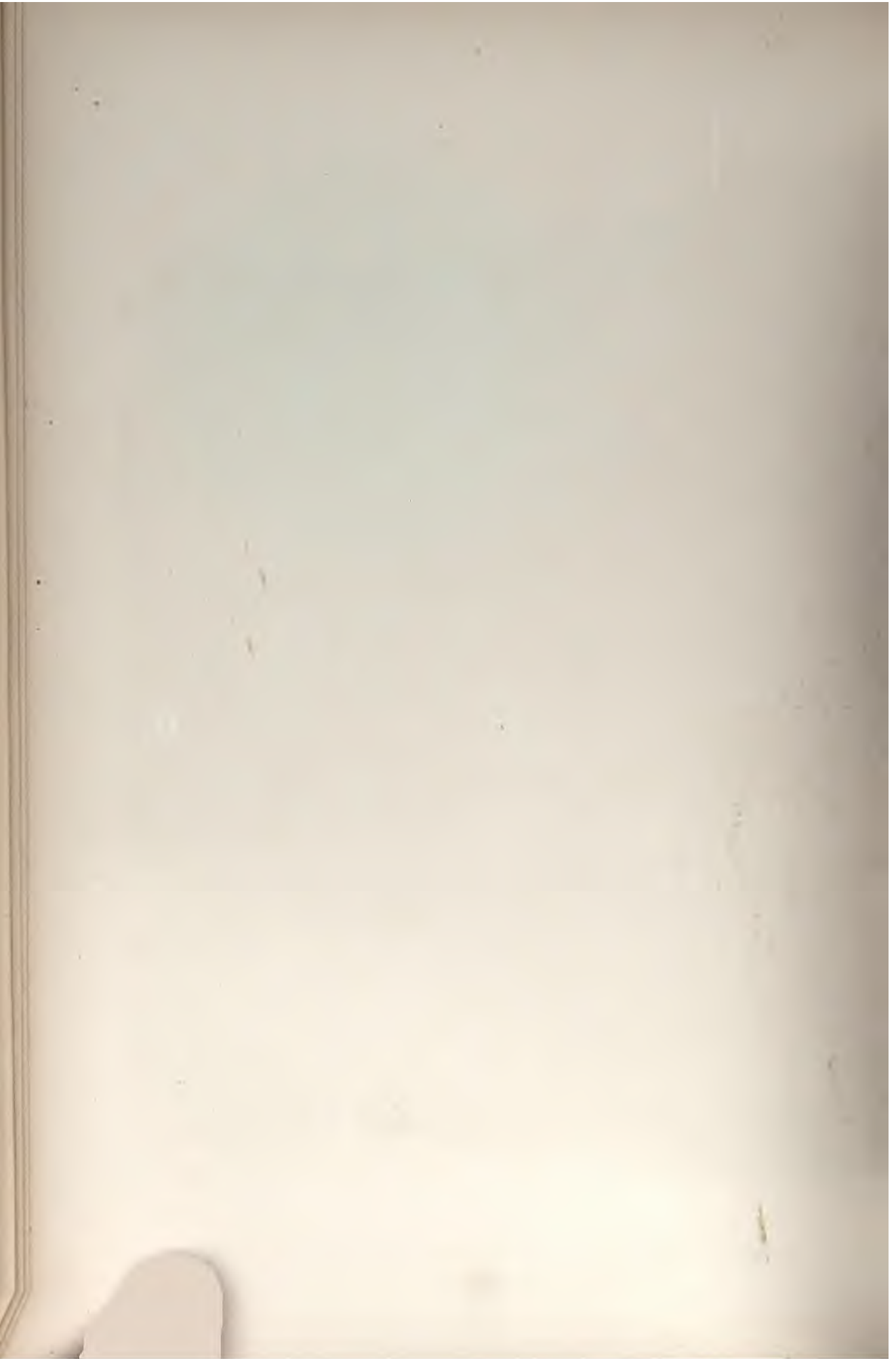
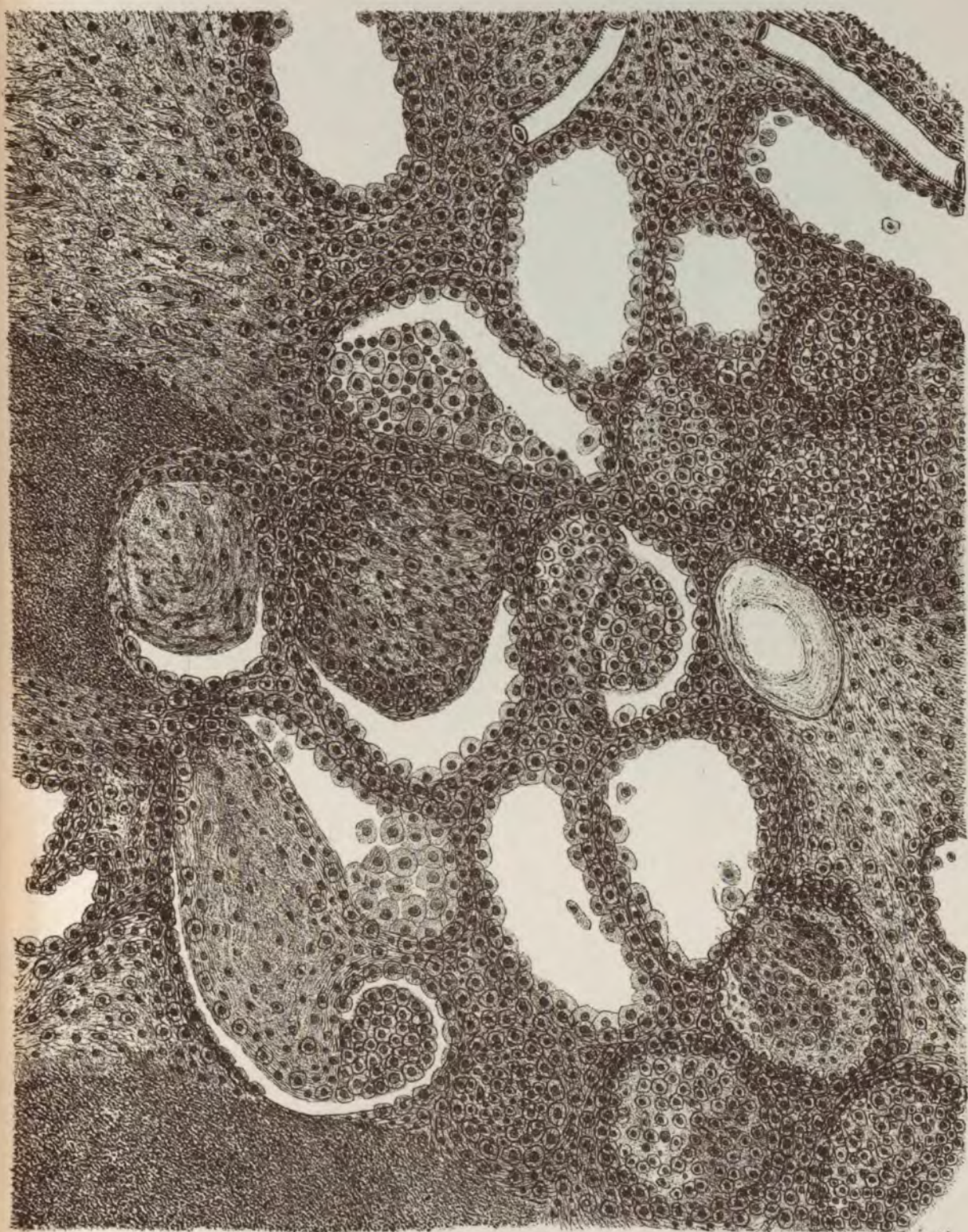


PLATE LIX.

*Tissue forming the periphery of a military Tubercle,
magnified 850 diameters.*

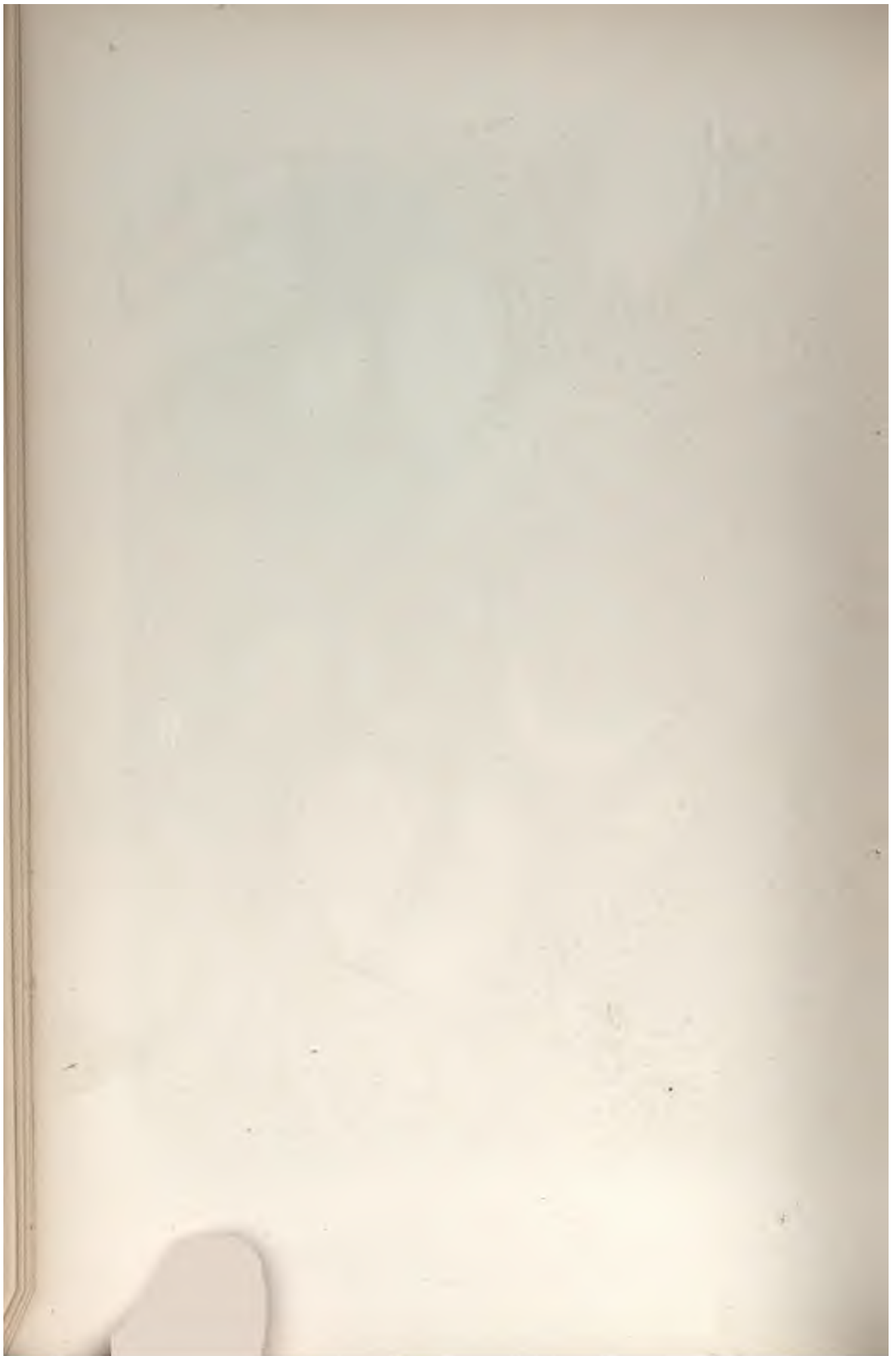


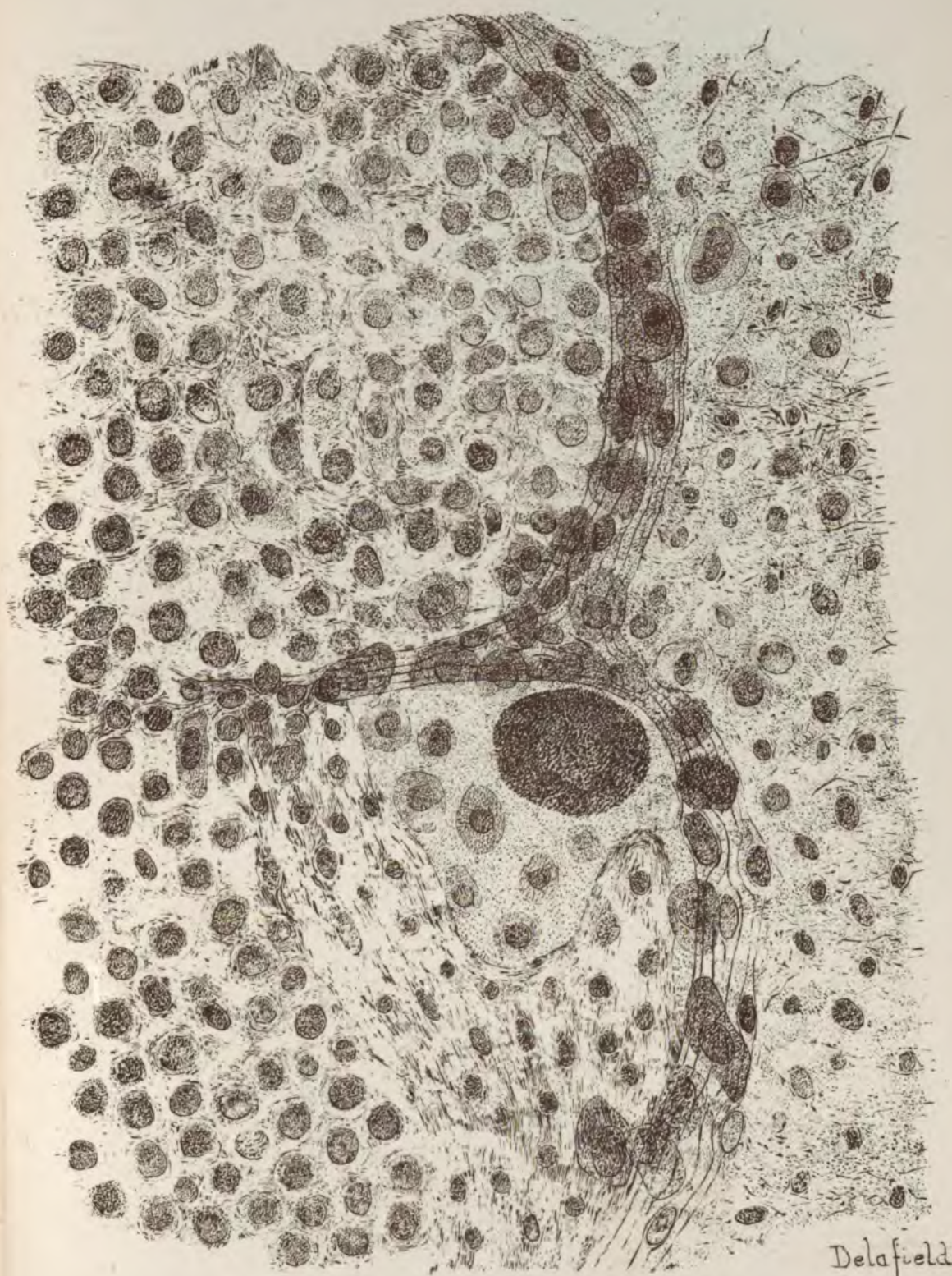


Delefield.

PLATE IV.

*Air Vesicles between Tubercles,
magnified 300 diameters.*



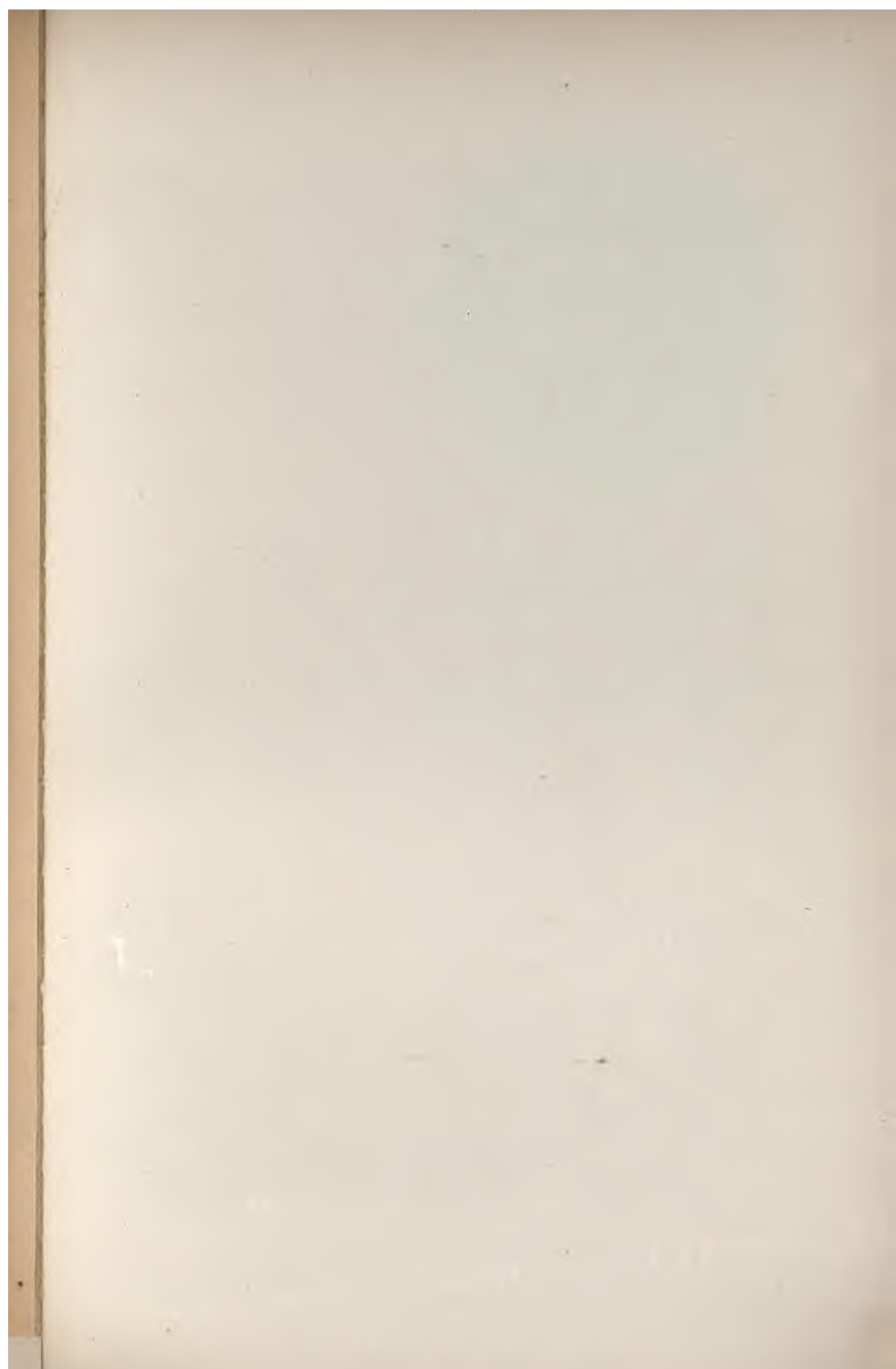


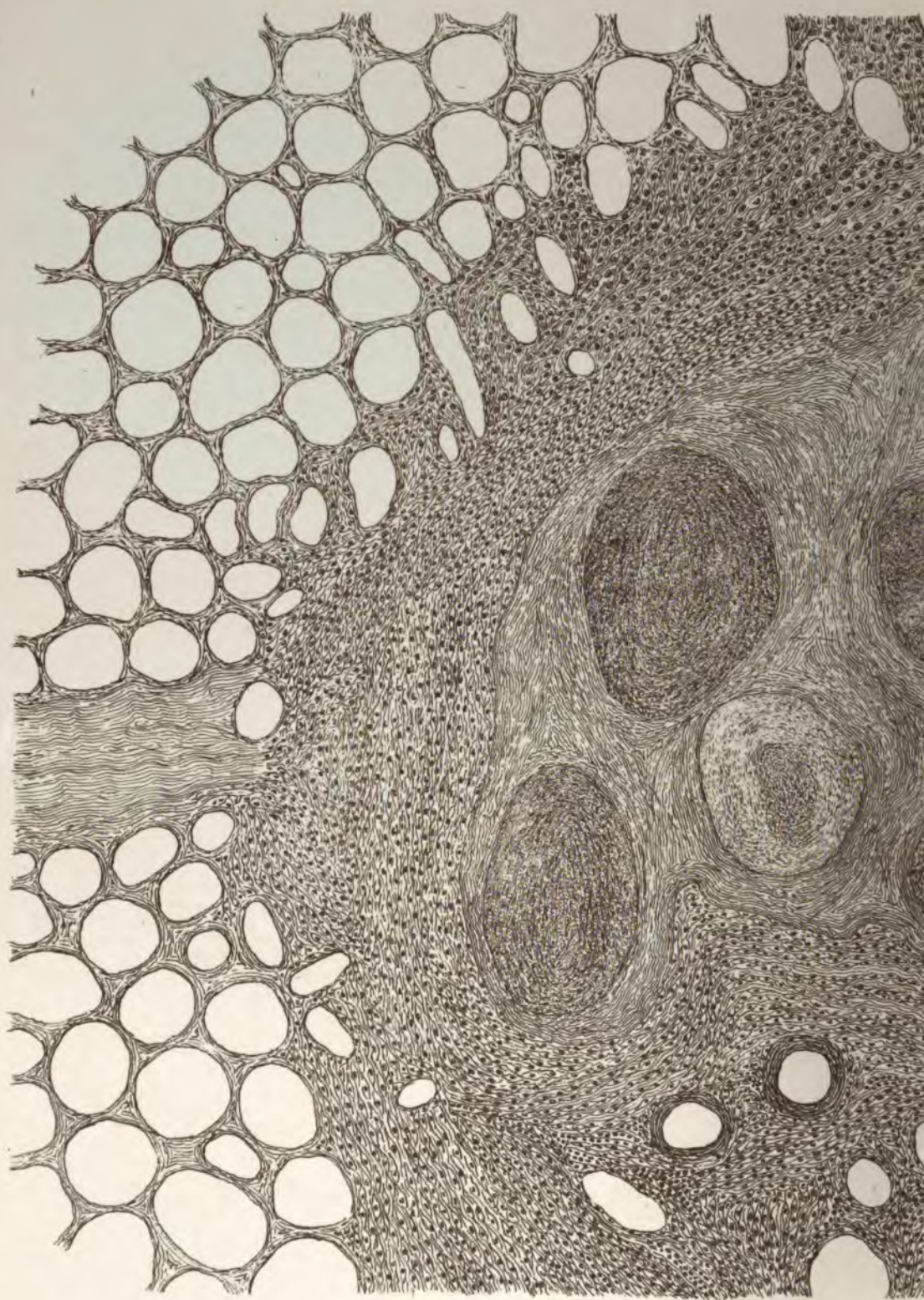
Delafield

PLATE LX.

*Air Vesicles from the periphery of a Tubercle,
magnified 850 diameters.*

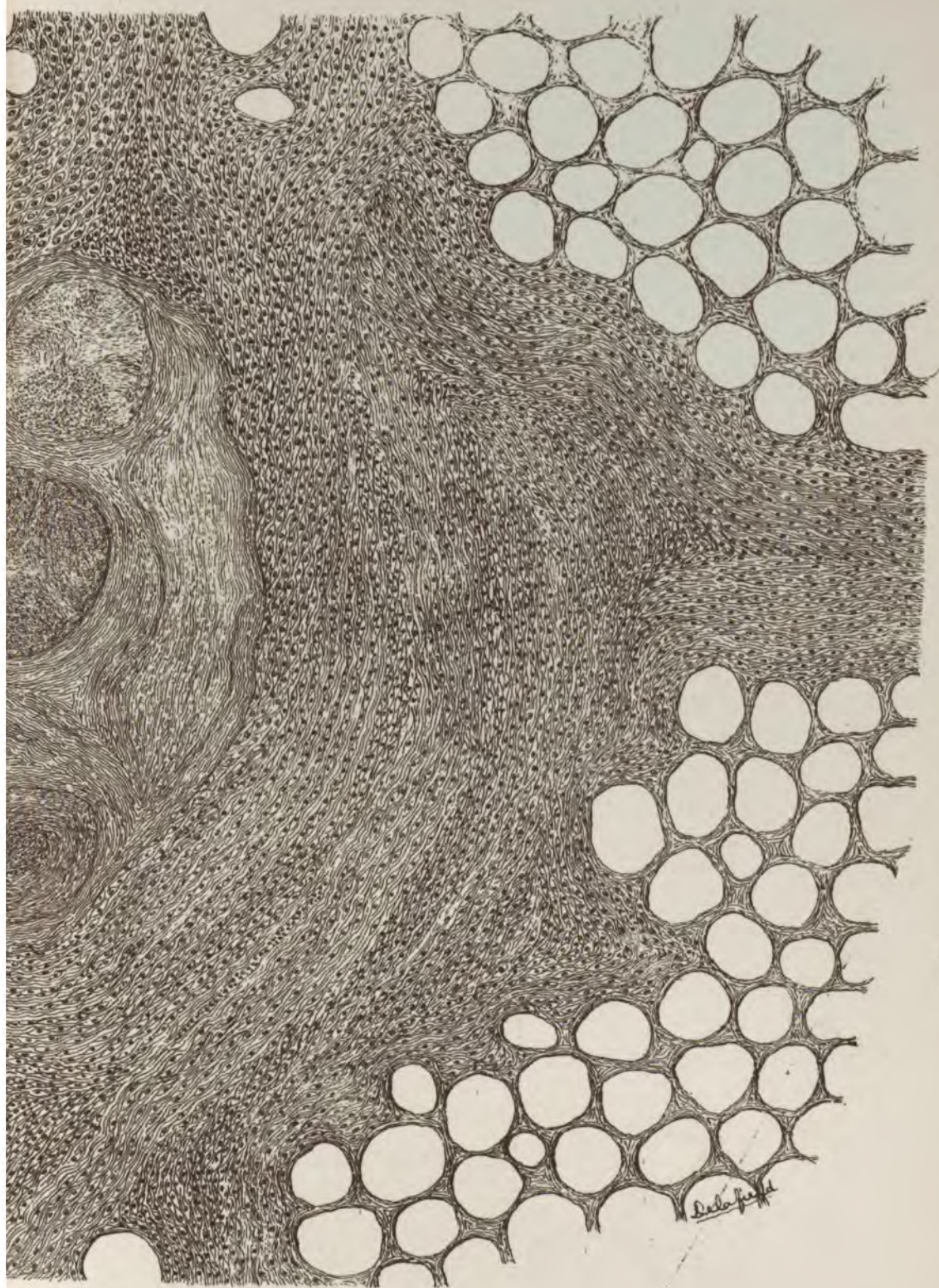






PLATE

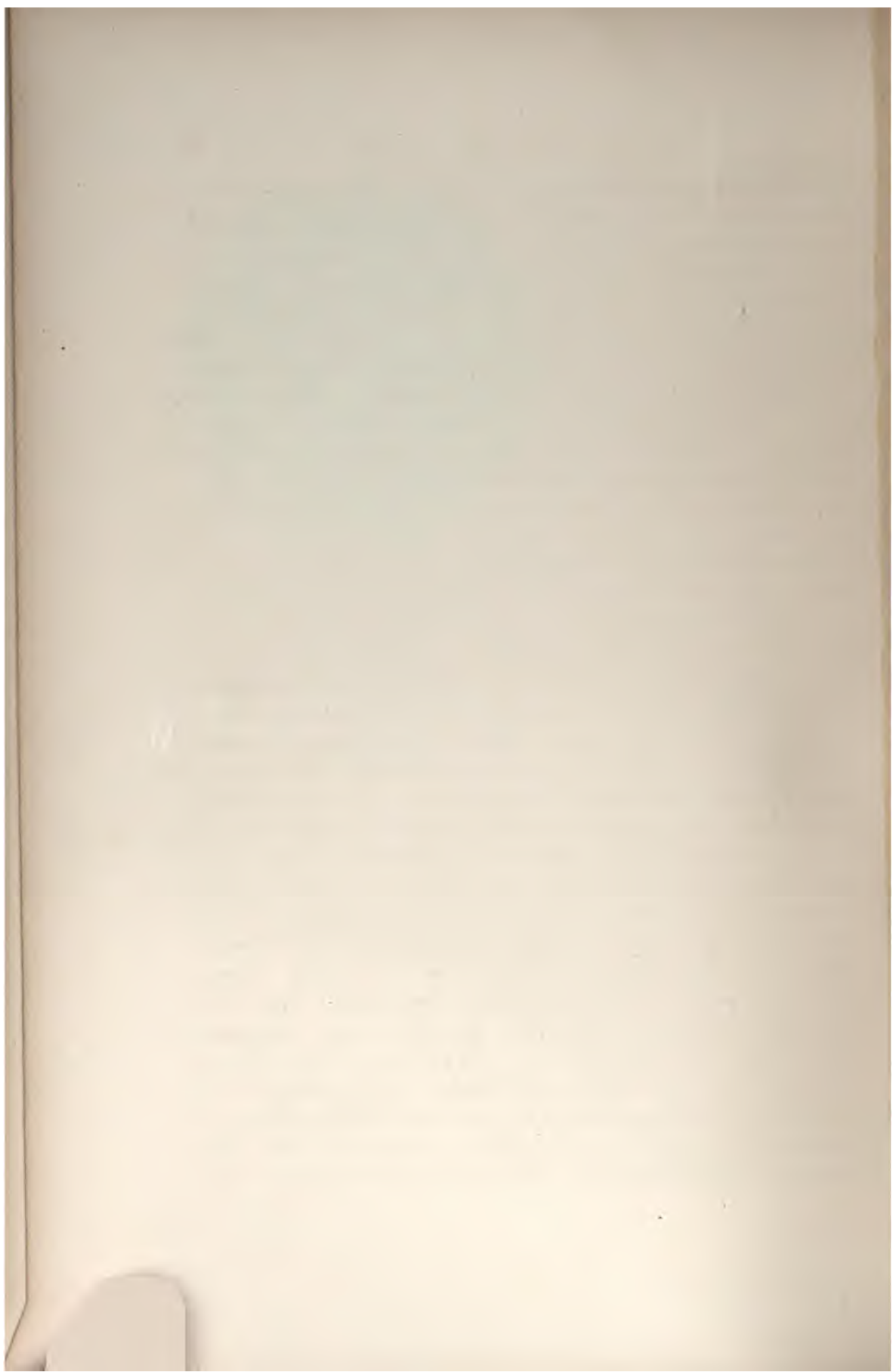
*A military Tubercle, changed
magnified 90*



Delo-feld

K.

*into fibrous tissue,
etc.*



tubercles appears, with a low magnifying power, like granulation tissue. Higher powers, however, show that its structure is more complex. It may really resemble granulation tissue as in Plate LIX.; or it may be a more complicated tissue, such as is represented in Plates LV. and LX.

In Plate LV. are a number of air-vesicles of which the outlines can be seen. Some of these are empty, while their edges are covered with large epithelial cells. Other air-vesicles are filled partly with epithelial cells, partly with smaller polygonal cells imbedded in a basement substance. Other air-vesicles are more or less completely filled by polypoid growths which project into them. These polypoid growths are formed of polygonal cells, and oval and round nuclei imbedded in a delicate basement substance. The portion of the air-vesicles which is not filled with such polypoid growths may contain epithelial cells.

Plate LX. represents portions of two air-vesicles more highly magnified. The walls of the vesicles are overlaid with large cells. At one point the wall splits up and its outlines are lost. One of the vesicles is filled with polygonal cells imbedded in a basement substance. The other vesicle is partly filled with similar tissue, partly with a denser, polypoid growth, partly with free epithelial cells and granular matter.

(3.) Nodules formed of tubercle-tissue in a condition of degeneration. The degeneration is of such a character that a larger or smaller part of the tubercles is converted into fibrous tissue, or granular matter, or both these changes may involve different parts of the same tubercle.

If the tubercle is changed into fibrous tissue, the centre of the nodule is formed of connective tissue arranged in a concentric shape, and imbedded in this are tubercle-granula also changed into connective tissue, or the granula may be changed into granular matter. Around this central portion is a zone of fibro-cellular tissue. Such tubercles may be very large, some of them are pigmented. Plate LXI. represents such a large miliary tubercle converted into fibrous tissue. Such tubercles as these are not sections of bronchi with thickened walls.

If the tubercles undergo cheesy degeneration, the portions first involved are the tubercle-granula. From these the degenerative process may extend and involve the diffuse tubercle in which the granula are imbedded. These tubercles are usually possessed of a peripheric zone

of fibro-cellular tissue composed of granulation tissue and altered air-vesicles, such as have already been described. Plate LIV. represents several miliary tubercles of which the tubercle-granula have undergone cheesy degeneration.

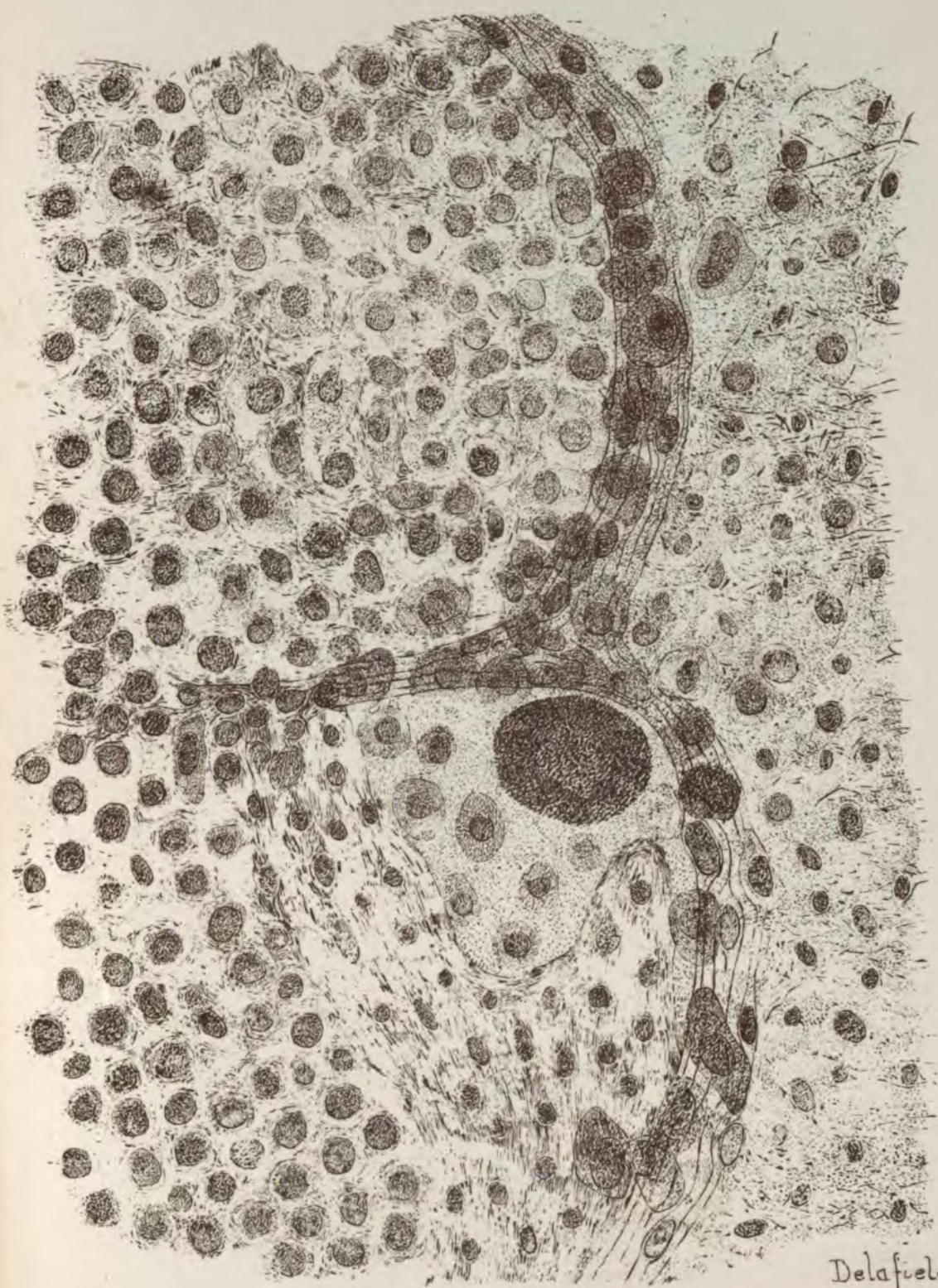
In a considerable number of cases of chronic miliary tuberculosis an important part of the pathological process consists in the development of the diffuse, solid tissue which forms the peripheric zone of some tubercles, which joins others together, which aggregates others into masses, and which may replace a considerable part of the parenchyma of the lung by a dense, solid, unaërated structure. In some cases, indeed, this diffuse tissue constitutes a more important part of the lesion than do the miliary tubercles, for it replaces and renders useless a larger part of the lung.

This diffuse tissue presents itself to us with a structure varying according to the length of time during which it has existed, and to understand it we must study both its earlier and later stages.

Even in the earlier stages the process is not a simple one, and we have to distinguish between a production of interstitial connective tissue with obliteration of the air-vesicles by pressure, and a growth of a peculiar new tissue within the cavities of the air-vesicles and in their walls.

The interstitial connective tissue is produced in the walls of the vesicles, the bronchi, and the blood-vessels, in the interlobular septa and the pulmonary pleura. Much of it is a dense, fibrillated tissue with but few cells, but well supplied with large, irregular blood-vessels. In other places the cells are more abundant, and in still others it looks like granulation tissue. It is often pigmented. The air-vesicles are deformed: some are small and misshapen, some are so compressed that they look like tubes; some are dilated. The epithelial cells which line the vesicles are increased in size and number. The whole process is essentially an extra-alveolar one, the vesicles suffering principally from pressure.

The growth of the tubercle-tissue is different. The process is both an extra- and intra-alveolar one. The solidification of the lung-tissue is accomplished by the formation of new tissue, both in the walls of the air-vesicles and within their cavities.

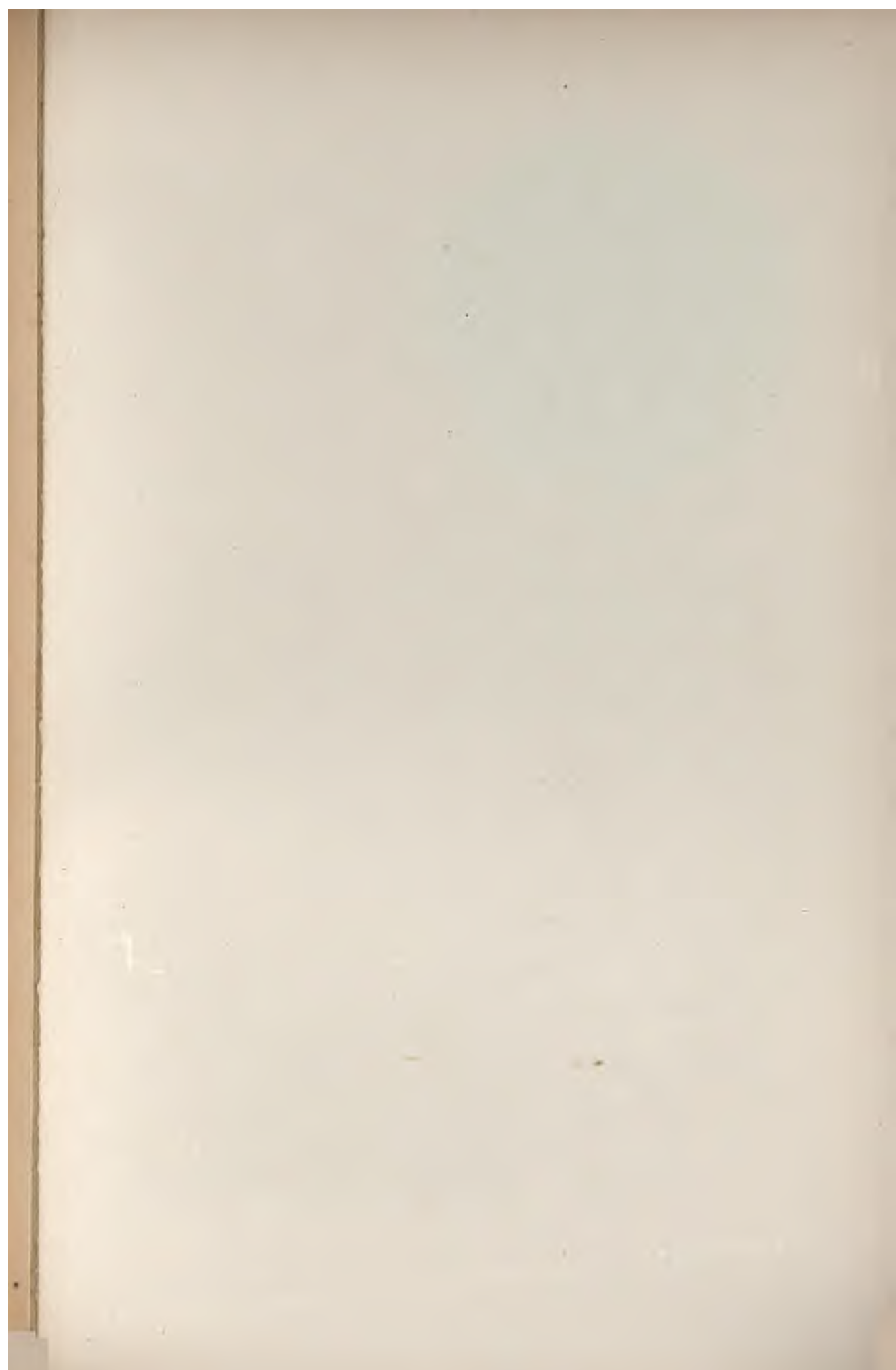


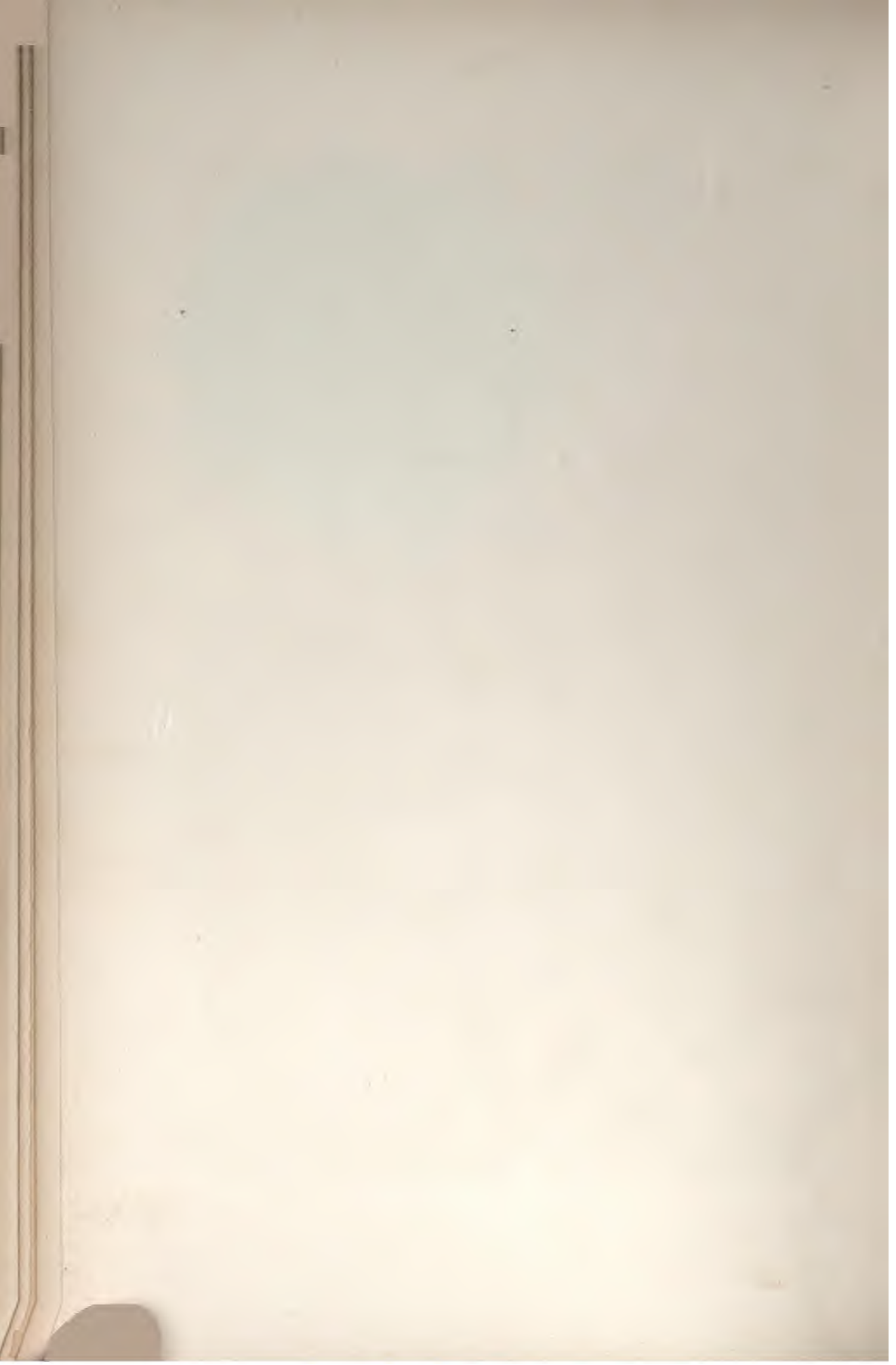
Delafield

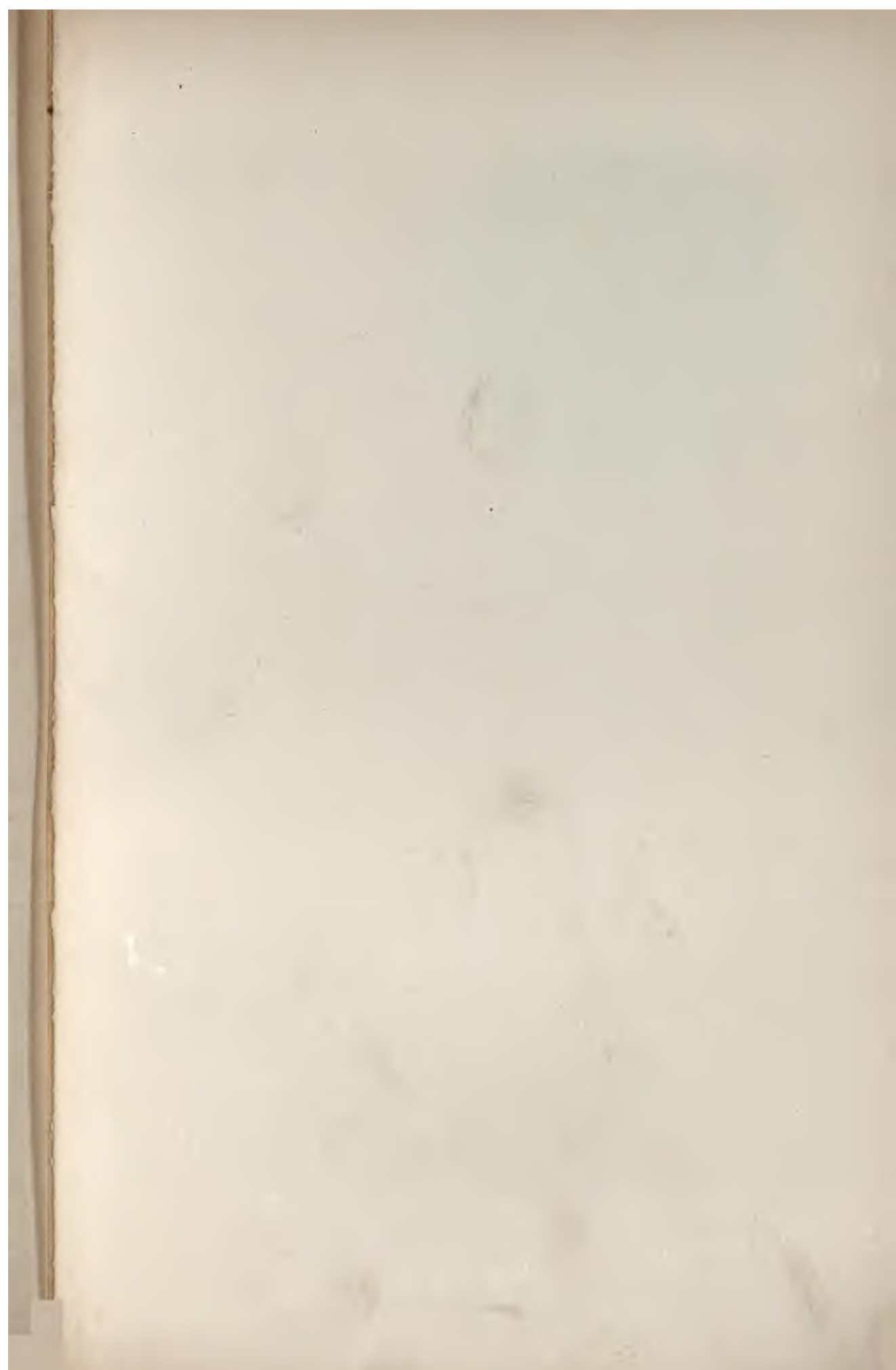
PLATE LX.

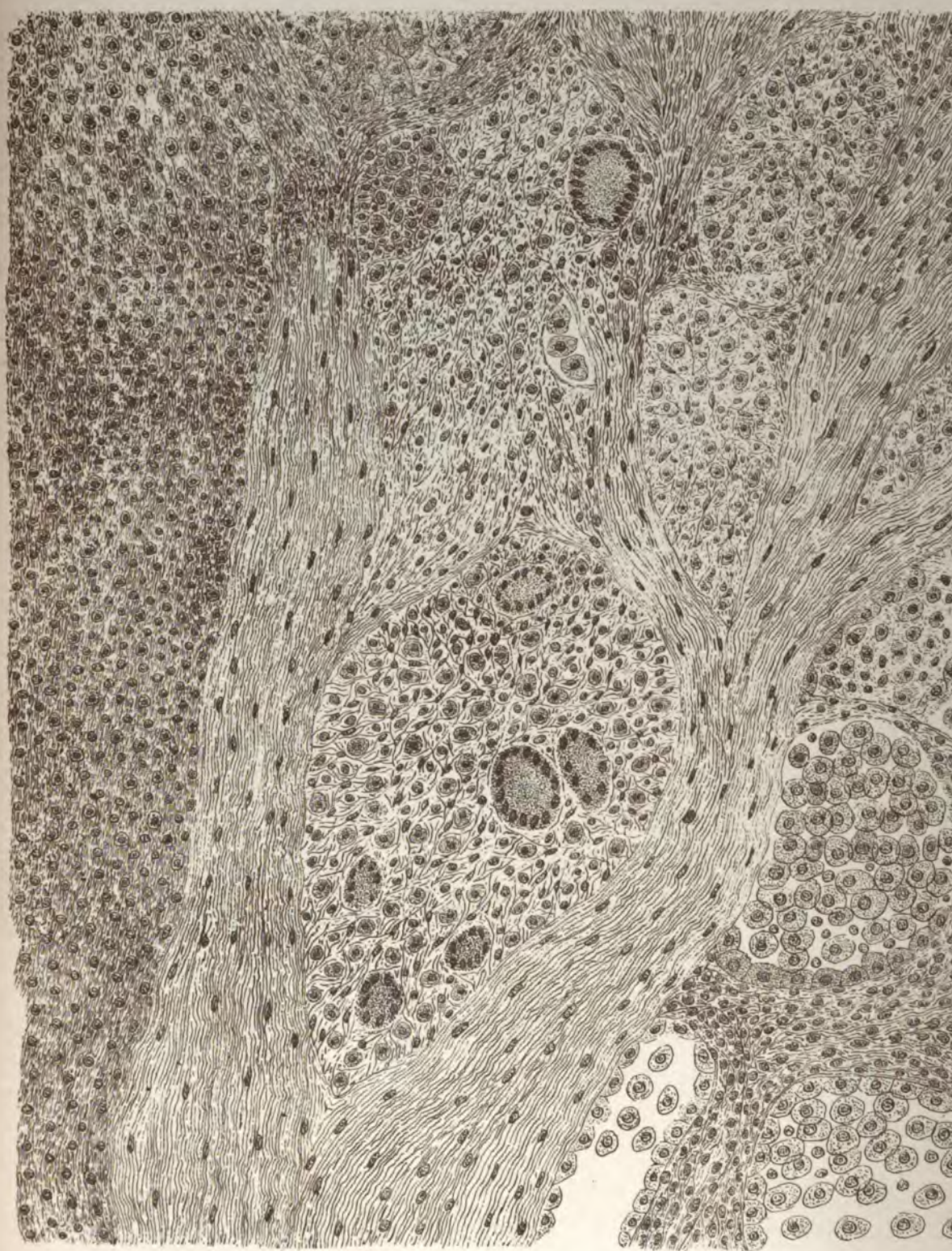
*Air Vesicles from the periphery of a Tubercle,
magnified 850 diameters.*





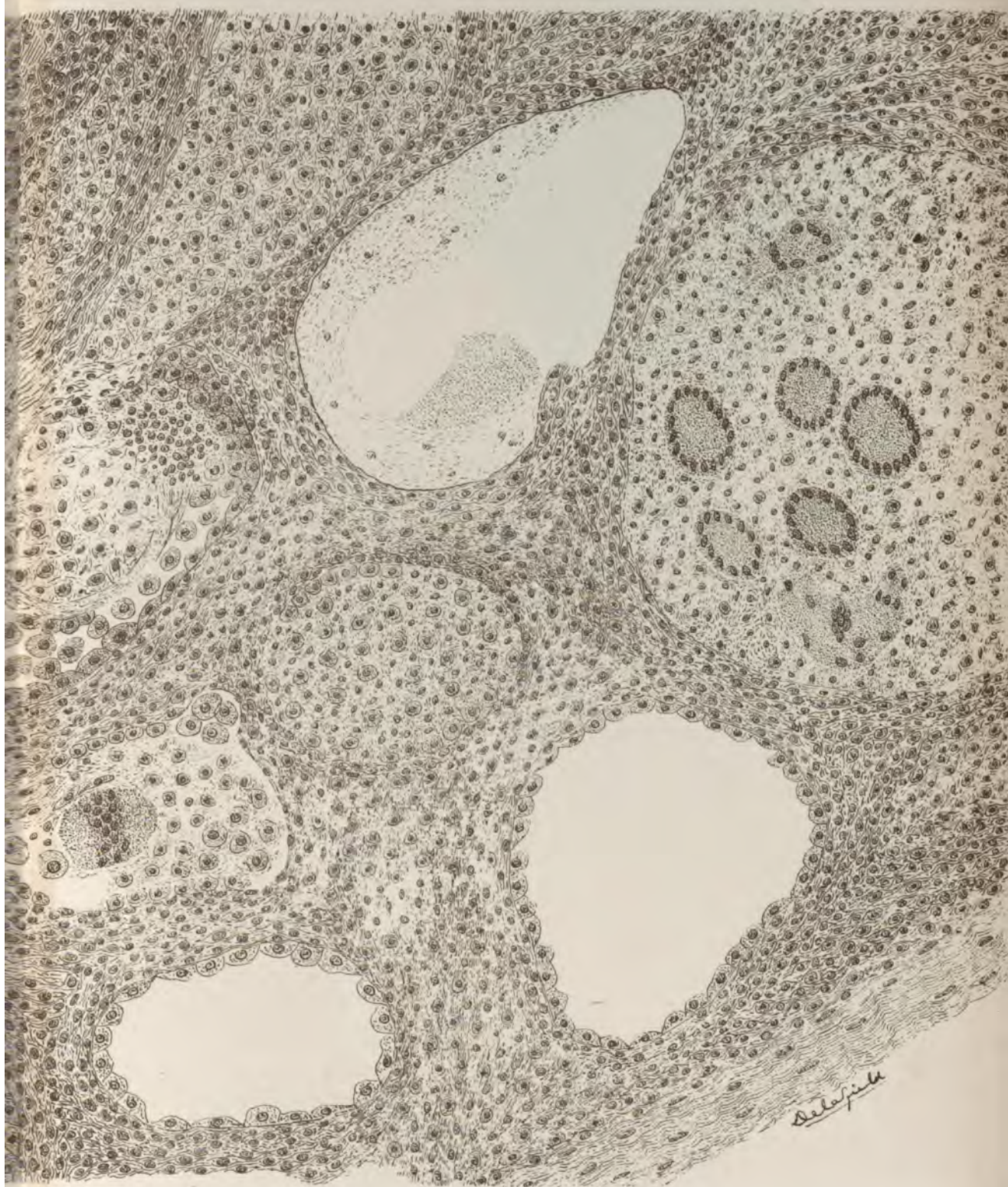






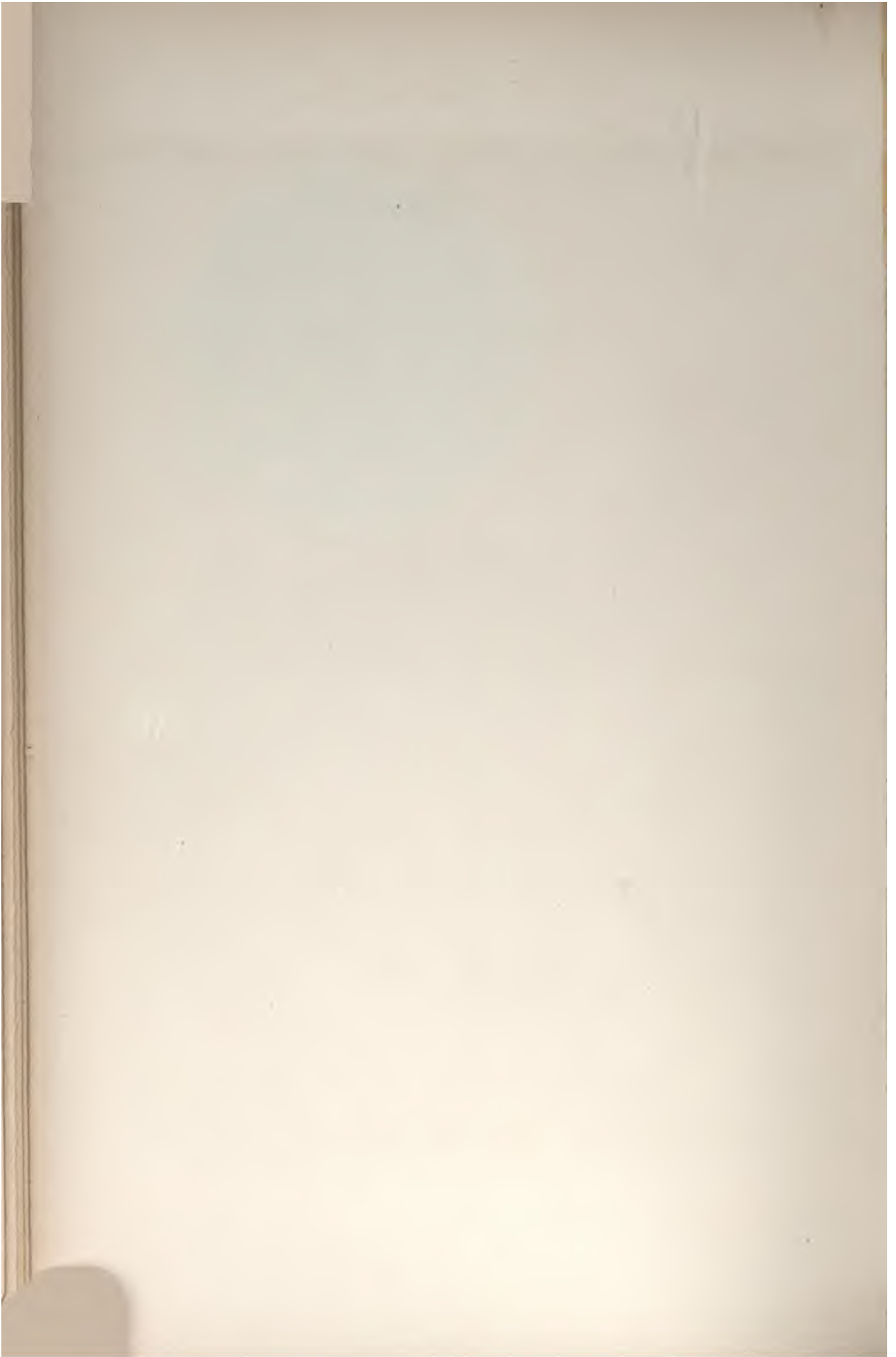
PLATE

*Diffuse
magnified*

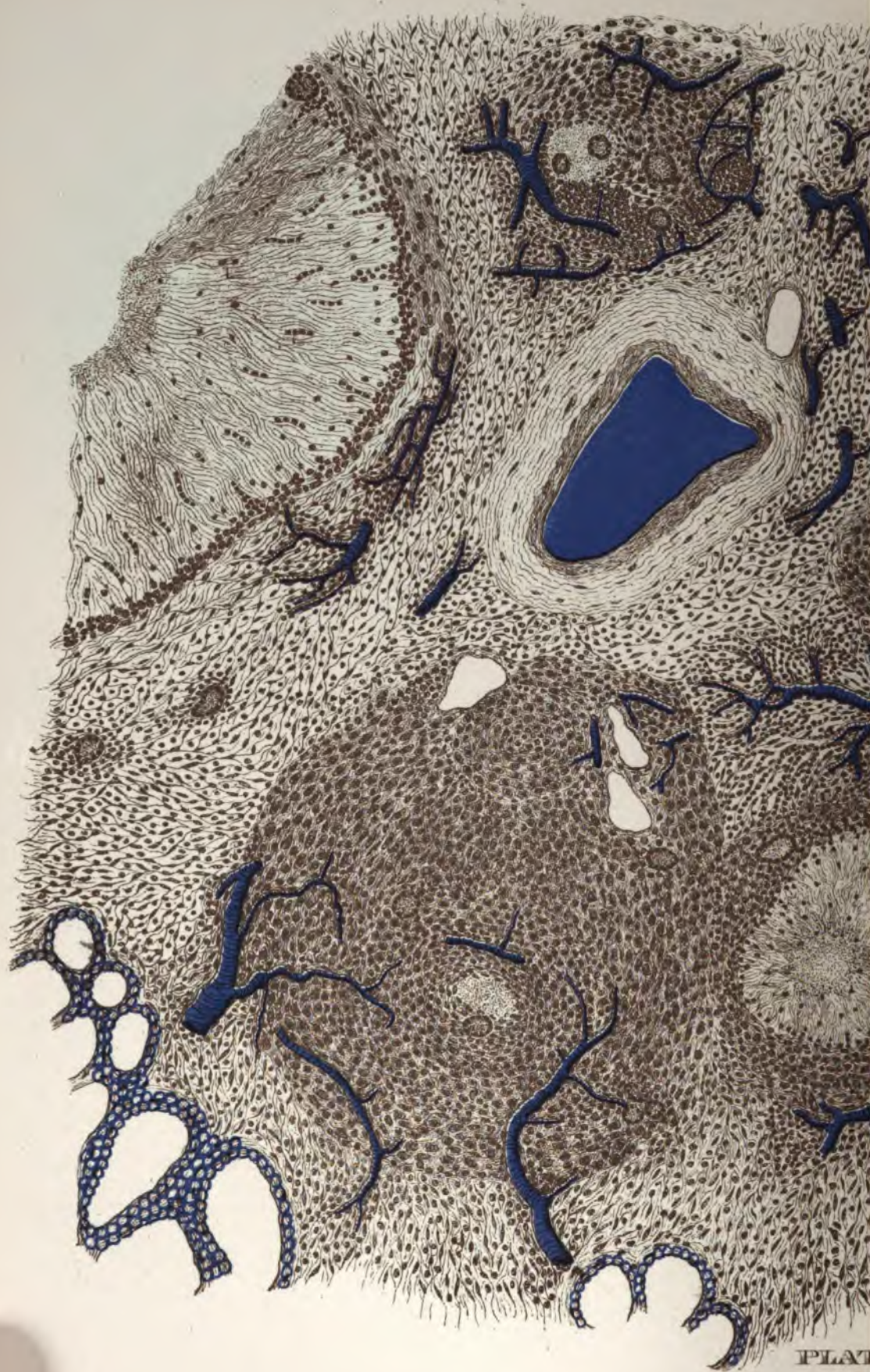


XXV.

Testis,
Leydig cells.

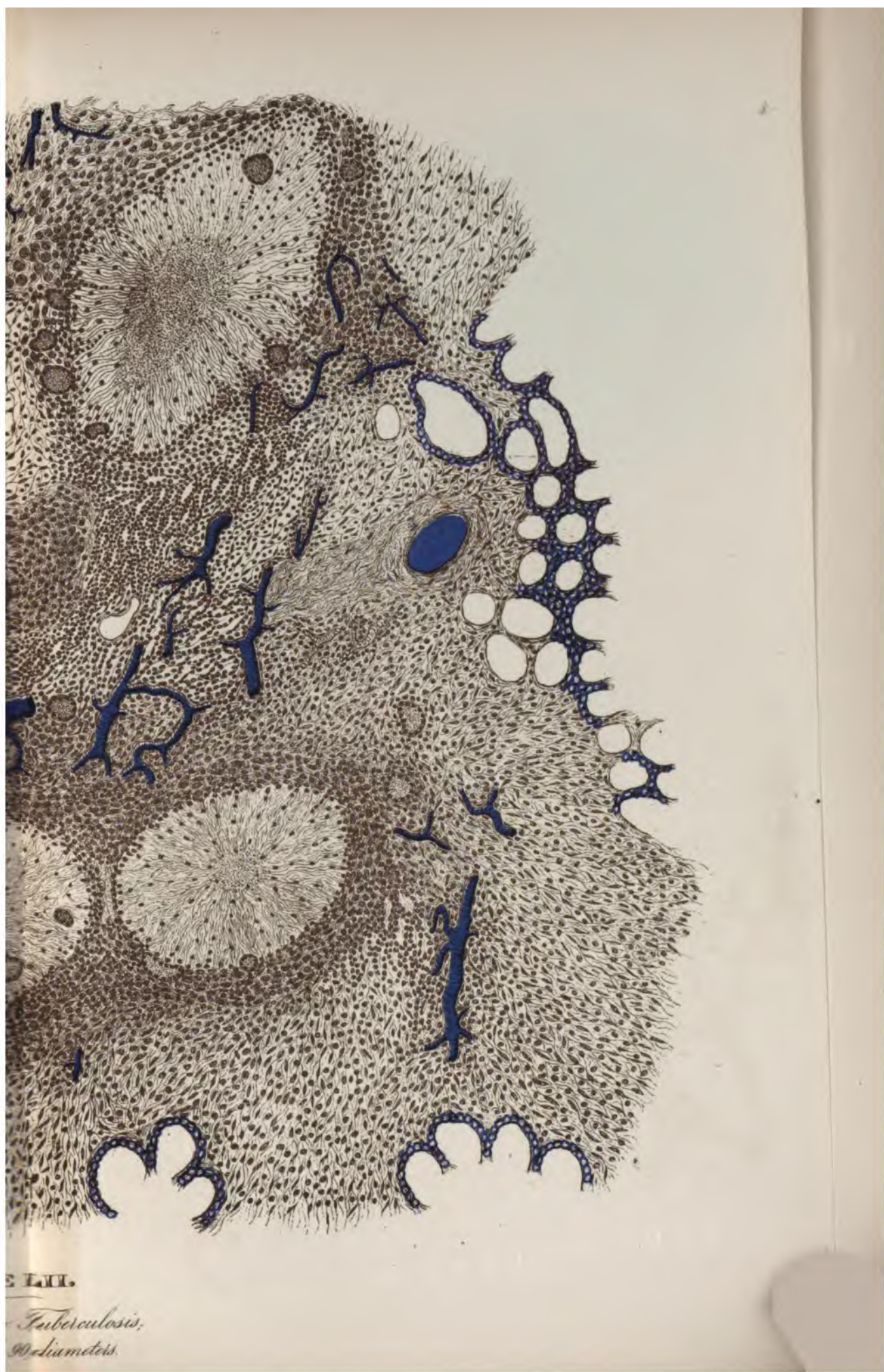






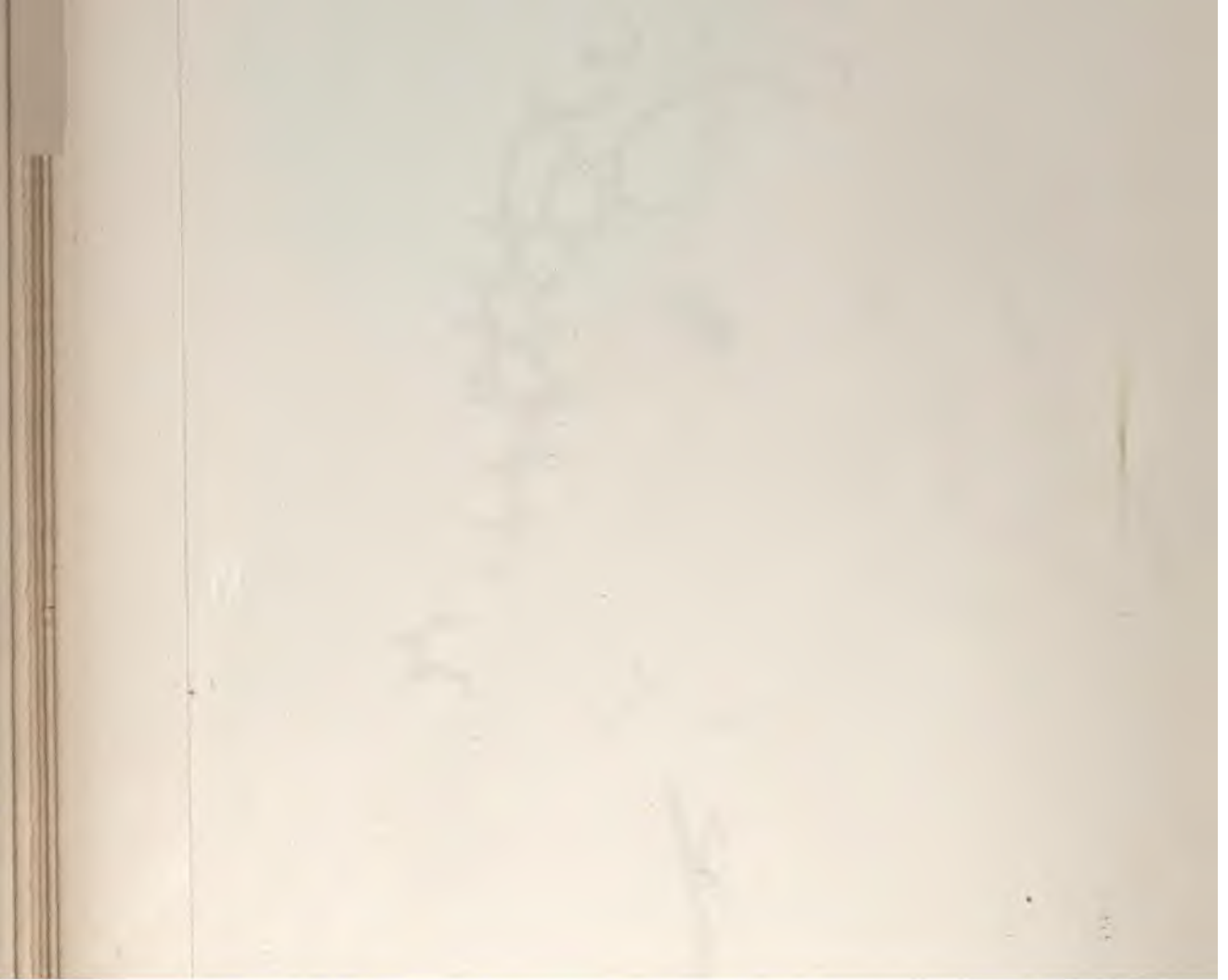
PLAT

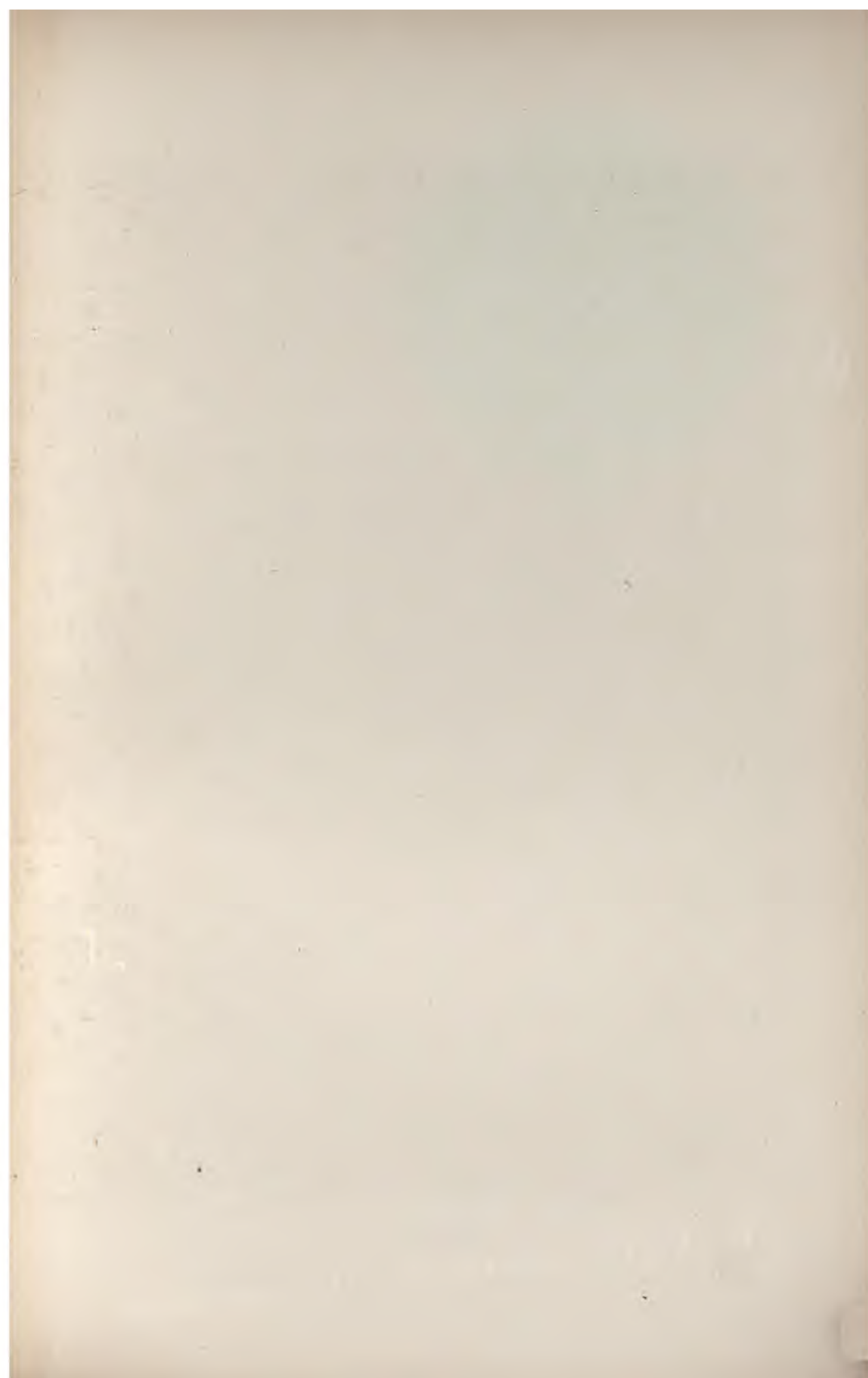
*Chronic miliar
magnifica*



E LII.

Tuberculosis.
90 diameters.





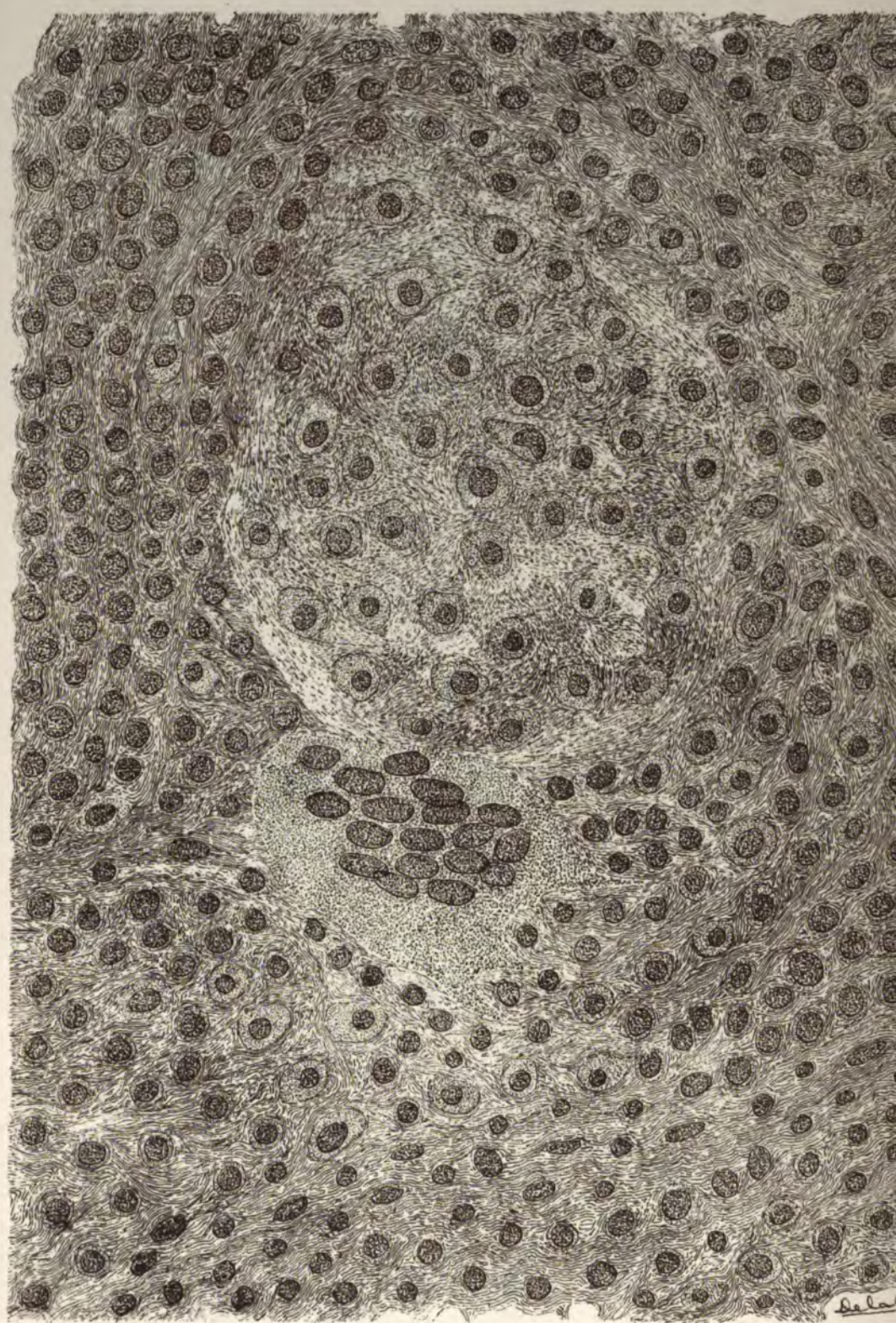


PLATE LVII.

*Tubercle Tissue between the miliary Tubercles;
magnified 850 diameters.*

In the walls of the air-vesicles there is a growth of round and polygonal cells and of intercellular basement substance, or the cells are formed in excess and the basement substance is split up so that the natural outlines of the wall of the vesicle are lost (Plate LX.).

The cavities of the vesicles are filled in two ways. Some are partly or completely filled with polypoid outgrowths from their walls, composed of round and polygonal cells imbedded in a basement substance; in others there is a mass of round and polygonal cells, with or without giant cells imbedded in a basement substance formed in the cavities of the vesicles, but at first not continuous with their walls. If the vesicles in either of these ways are not completely filled the remaining space may be occupied by epithelial cells. When an air-vesicle is completely filled with cell growth, it looks like a tubercle-granulum. Plate LV. shows the filling up of air-vesicles by polypoid outgrowths from their walls. Plate LXIV. shows a number of air-vesicles so filled as to look like tubercle-granula; in some places the vesicles have run together so that their outlines are lost. There is also an abundant interstitial growth of tubercle-tissue and connective tissue.

These two processes—the growth of interstitial connective tissue, and the growth of tubercle-tissue in the walls and in the cavities of the air-vesicles—are usually associated. When both have existed for some time the picture becomes a complicated one.

We can no longer make out the air-vesicles, nor the relations of the new tissue to them. There is nothing but a solid mass, composed of miliary tubercles surrounded by tubercle-granula, diffuse tubercle, connective tissue, and a few deformed air-vesicles. Plate LII. represents such a condition, the blood vessels having been artificially injected with blue. There are in the field three old, cheesy miliary tubercles and part of a fourth. Between these is a solid mass composed of connective tissue, diffuse tubercle and tubercle-granula. The blood-vessels are large and irregular, and some of them penetrate into the granula. Plate LVII. represents a single tubercle-granulum surrounded by diffuse tubercle from such a mass of old solid tissue. It seems probable that in these old formations tubercle-granula are formed not only in the cavities of the vesicles, but in the interstitial tissue.

When the disease is of still longer duration, the growth of new connective tissue becomes more marked, and a considerable part of the tubercle-tissue is changed into connective tissue. The affected portions of the lung then become very hard and dense, and we have the condition so often called fibrous phthisis. But such a fibrous phthisis is nothing but an old tubercular phthisis.

CONCLUSIONS.

1. There is a form of chronic pulmonary phthisis of which the characteristic lesion is the production of a new tissue in the parenchyma of the lungs.

2. This new tissue is not an ordinary inflammatory product, but resembles the tissue which composes most of the miliary tubercles in acute miliary tuberculosis.

3. This new tissue is arranged in the form of nodules—miliary tubercles, and in the form of a diffuse infiltration which surrounds the tubercles, joins them together, and may occupy considerable portions of the lung.

4. The tubercle-granula seem to be formed, for the most part, within the cavities of the air-vesicles.

5. Associated with the production of tubercle-tissue there is often interstitial pneumonia, bronchitis and pleurisy, with their characteristic inflammatory products.

6. The tubercle-tissue is often changed into granular matter, or into connective tissue. In old cases of the disease the development of the different elements of the lesion can no longer be made out. There is simply a mass of tubercle-tissue, more or less degenerated, and of connective tissue.

7. The disease runs a chronic and progressive course, gradually involving more and more of the lung-tissue. But it may be arrested and the patient may recover permanently.

8. Tubercle-tissue may also be formed in other parts of the body especially in the larynx, small intestine and peritoneum.

ACUTE PHTHISIS.

THE clinical characters of acute phthisis are sufficiently well known. A person who has previously been in good health, or who has already suffered from chronic phthisis, either suddenly, or after a few days of indisposition, becomes seriously ill. There is a well-marked febrile movement and a rapid pulse, cough, expectoration, the physical signs of consolidation of the lung, and sometimes hæmoptysis. Rapid loss of flesh and of strength follow. Most of the patients die at the end of a few weeks or months; a few recover from the immediate attack and go on to have the history of chronic phthisis.

After death we find a considerable portion of one or both lungs consolidated, but this consolidation is not of a uniform appearance. The larger part of the consolidation looks like ordinary red hepatization, or else it is of a peculiar grayish color, different from the gray hepatization of lobar pneumonia. In this diffuse hepatization are other portions of solidified lung of different appearance. They are of a white or yellow color, much denser than the surrounding hepatization, or they may be softened at their centres. These white solidifications are of many different sizes and shapes. The smallest are hardly larger than an air-vesicle, the largest may include a considerable part of a lobe. Their size and shape are made more evident if the blood-vessels are artificially injected, for into these nodules the injection does not run.

Many of the smallest of these whitish masses are scattered irregularly through the lung-tissue like miliary tubercles. As will be seen later, they really are miliary tubercles.

Other similar masses, also of small size, are situated regularly around small bronchi. When this is the case, the cavities of the bronchi may be filled with inflammatory products, and their walls infiltrated.

Still other masses will correspond to a single air-passage, or to a group of air-passages, looking as if they were produced by some cause which affected definite and isolated portions of the lung.

Other masses again are so large that they have the appearance of a diffuse infiltration of a considerable portion of the lung. But these large infiltrations seem to be made up of a number of smaller masses.

If the patient lives long enough after the development of the disease, many of these masses, both large and small, will be found degenerated and softened.

The bronchi of the affected lung are regularly the seat of morbid changes. They may be congested and coated with mucus; they may be filled with pus, epithelium, and fibrine, and these inflammatory products may undergo cheesy degeneration; they may be irregularly dilated, so as to form cavities of some size; or a great number of the smaller bronchi may be symmetrically dilated, so that on section the lung looks honeycombed.

Such a condition of the lung has been interpreted in very different ways.

Some look upon the whole process as one of ordinary inflammation. Bronchitis, peri-bronchitis, ulceration of the bronchi, and pneumonia with subsequent degeneration of the products of inflammation are supposed to produce all the lesions.

Others believe all the white or yellow masses to be of essentially the same nature as miliary tubercles, that they are tubercles on a large scale "tubercules massifs;" and are surrounded by ordinary pneumonic hepatization. Others again regard the whole conditions as a combination of pneumonia and miliary tubercles.

Neither of these views seem to me to account for all the lesions.

The pathological condition is really a complex one, and we gain nothing by trying to make it appear more simple than it really is.

It is necessary to examine each of the different parts of the lesion in detail and see what their real structure is.

I. The diffuse hepatization.

It must be remembered that we are considering the diffuse hepatization of acute phthisis, not that of chronic phthisis, and especially the conditions found in the lungs of adults. This hepatization presents the gross appearance of the red hepatization of lobar pneumonia, except that it is smoother; or it is of a peculiar gray color, sometimes almost gelatinous looking. It does not seem to degenerate or to soften except immediately around some of the nodules of which we shall speak presently.

The blood-vessels in the walls of the air-vesicles can be easily filled with an artificial injection. The walls of the air-vesicles show no marked changes in their substance, although their surfaces are overlaid with new cells.

The cavities of the air-vesicles are filled with inflammatory products, but this filling is not uniform. Some vesicles are filled and even distended, while others only contain a small amount of such new substances. These inflammatory products are: pus-cells, fibrillated fibrine, large, polygonal, nucleated cells resembling epithelium, minute, shining granules and a peculiar transparent matter. The pus-cells have the ordinary appearance of these bodies, varying in their size and in the character of their cell-substance, some being finely, some coarsely granular, and some fatty. The fibrine has the same appearance of a network of fibrils as is seen in acute, lobar pneumonia. The large polygonal cells resemble, in shape and appearance, epithelial cells, but some of them undergo degenerative changes, becoming larger, globular, coarsely granular or fatty, or very large and transparent. The granules are small, shining particles, not dissolved by acetic acid, liquor potassæ or chloroform, and are stained either with methyl violet or hæmatoxylin. Their nature is uncertain. These different products are found in variable relative proportion in different lungs and in different

parts of the same lung. Some vesicles will be entirely filled with the granules, others with fibrine, others with epithelial cells; while others will contain granules and epithelium, or epithelium and fibrine, with an admixture of pus-cells.

Plate LXV. represents a single air-vesicle, its cavity filled with large epithelial cells and a few pus-cells, the vessels in its walls injected. Plate LXVI. represents a single vesicle filled with coagulated fibrine, in which are entangled a few pus-cells and epithelial cells; the blood-vessels are injected. Plate LXVII. represents a group of vesicles filled with granules and a few epithelial cells; the blood-vessels are injected.

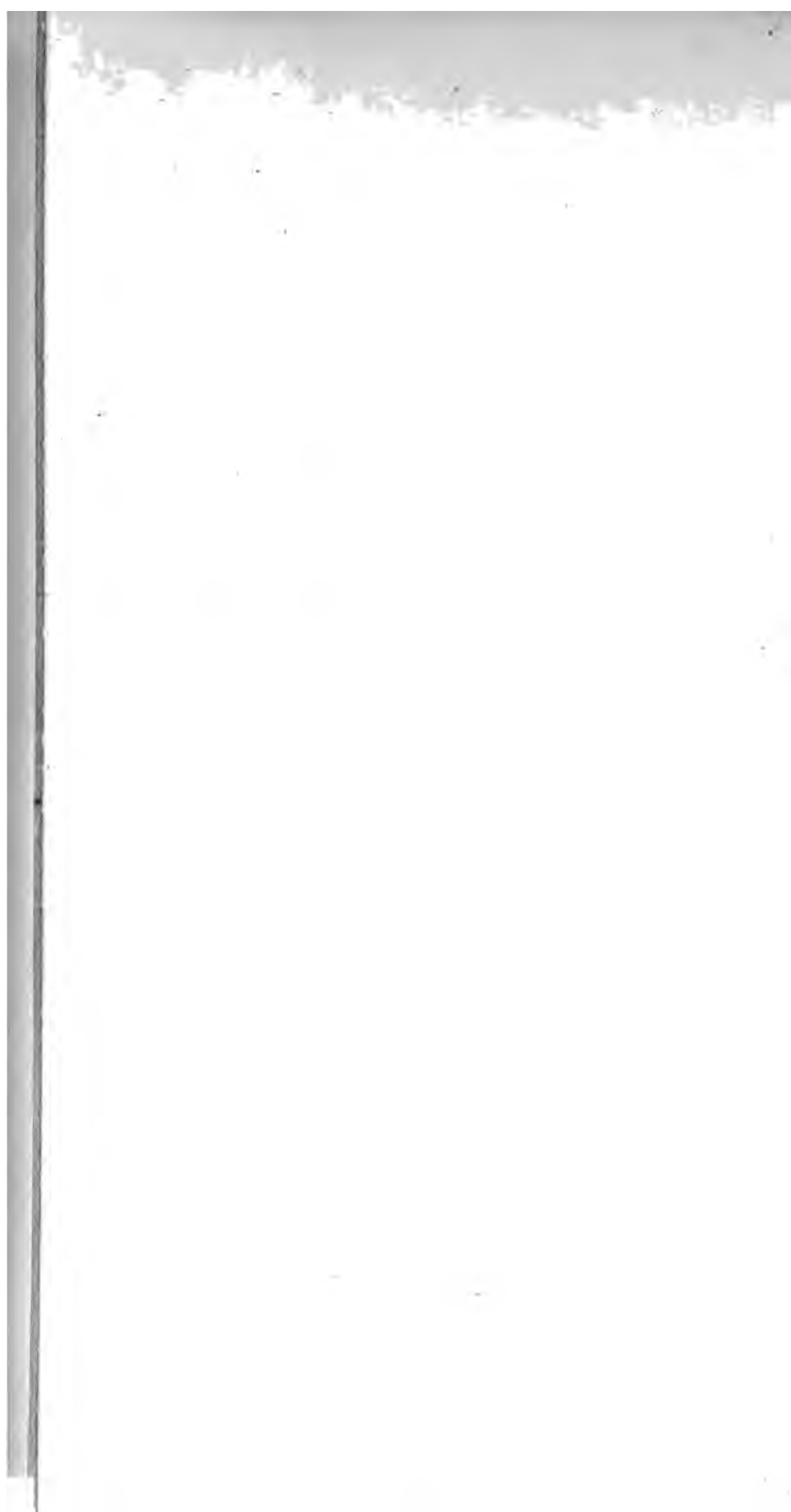
This diffuse hepatization seems to have no tendency to cheesy degeneration or necrosis. The products of inflammation within the vesicles may undergo fatty degeneration, but the walls of the vesicles remain intact, and the blood-vessels pervious. If we can judge from clinical experience and physical signs, this diffuse hepatization is capable of resolution and may entirely disappear. If, however, the patient goes on to have chronic phthisis then the diffuse hepatization undergoes marked changes.

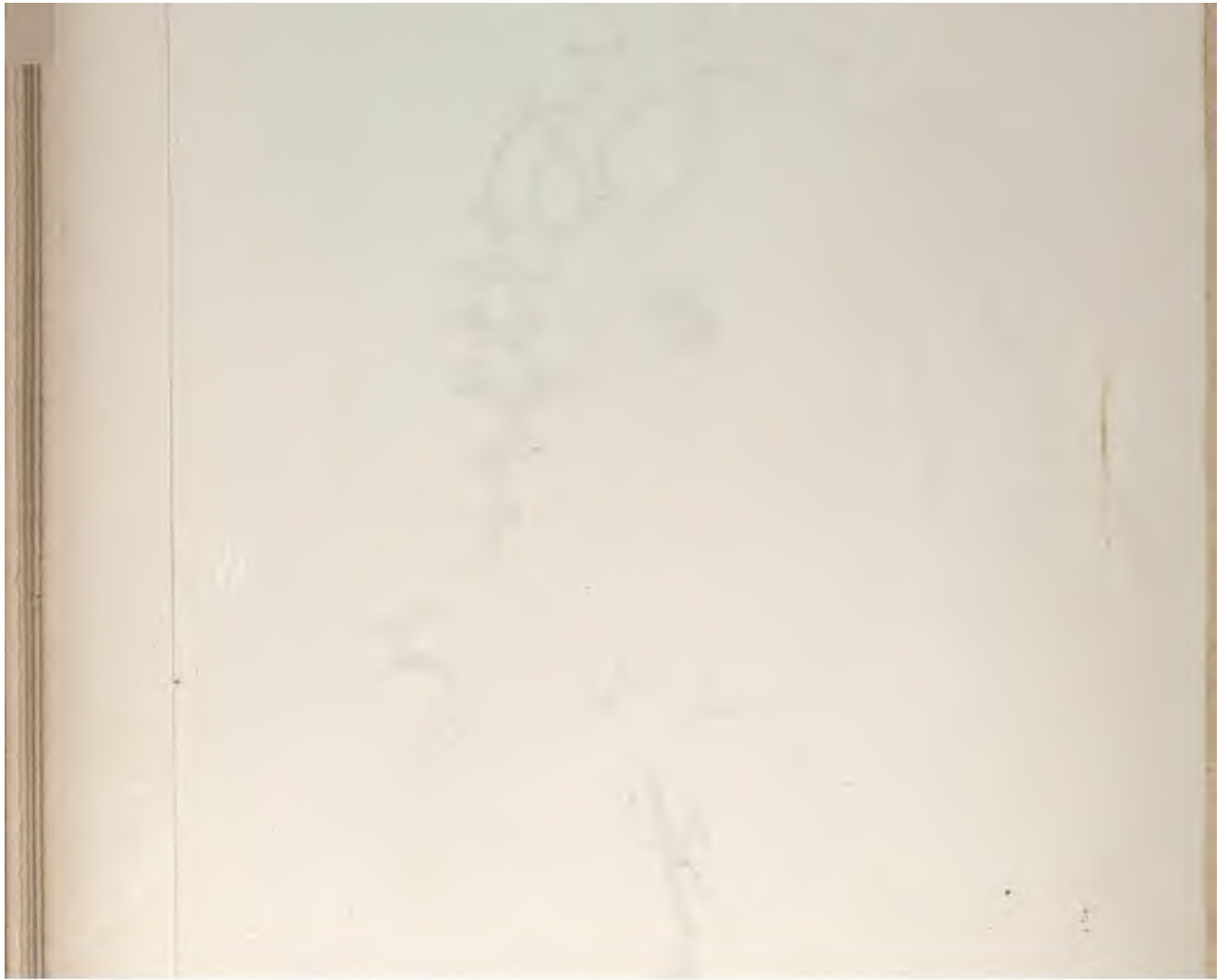
II. The white or yellow nodules.

These nodules vary in their size, their shape, and their structure. They may be close together, or widely separated. Some are surrounded by diffuse hepatization, others by nearly normal lung. Even those which present the same appearance to the naked eye are not all of the same nature.

Before attempting to describe these nodules it is necessary to have a definite idea of the arrangement of the small bronchi and air-vesicles.

Quain's Anatomy gives the following description: "The small bronchial tube entering a lobule divides and subdivides a variable number of times, according to the size of the lobule; its divisions losing their cylindrical form, and being converted into irregular lobular passages, are beset, at first sparingly, but afterward closely and on all sides, with numerous little recesses or dilatations, and ultimately terminate near the surface of the lobule in a group of similar recesses.









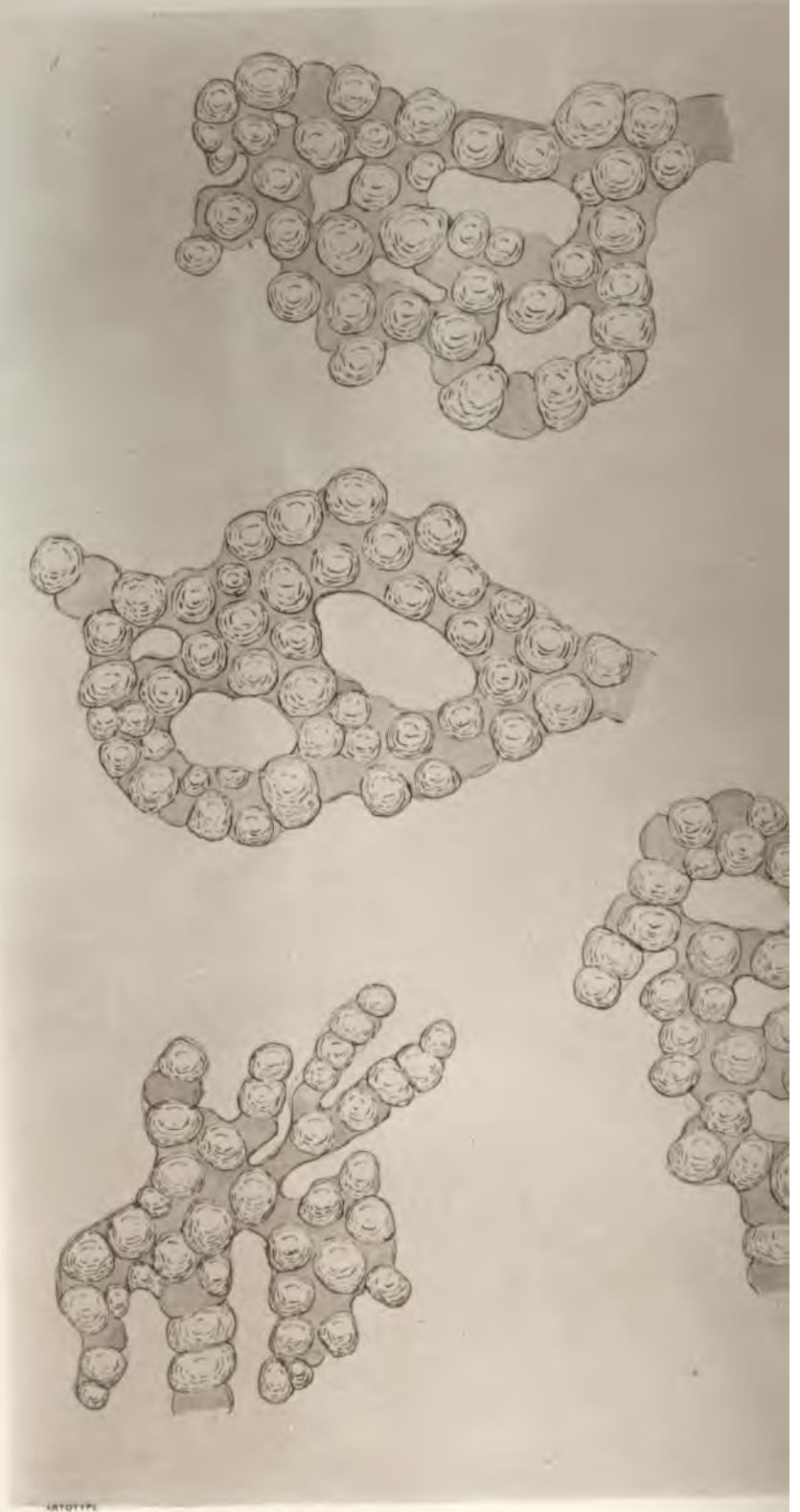
that the lung is made up of lobules; but that while on some of their sides the lobules are separated from each other by fibrous septa, on other sides they are continuous with each other.

When the corroded lung is broken up into lobules it is seen that the bronchi enters the lobules irregularly. Some bronchi enter the lobules at their ends nearest the root of the lung; others at the side of the lobules; others run along the side of a lobule and send branches into it at right angles.

When the lobules are broken up into fragments, we see such figures as are represented in Plate LXVIII. Here are fragments of a bronchiole, and of several air-passages, as seen by reflected light. The air-passages seem to be made up of a succession of large vesicles opening into each other, or of an irregular, larger canal, made up of vesicles, into which other vesicles open from all sides. These air-passages branch and anastomose as seen in the drawing. They are given off from the ends of terminal bronchioles or from the sides of small bronchi. The structure of the walls of the air-passages is the same as that of the air-vesicles. Each lobule seems to consist principally of such air-passages; the bronchi and air-vesicles proper do not constitute as large a part of the parenchyma as do the air-passages. If we look at the air-passages shown in the plate, and imagine them to be cut in different directions, it will be seen what a variety of figures the cut surfaces will give. If we make rather a thick section of an injected lung, and photograph this section with a low power, as seen in Plate LXIX., we can get an idea of the shapes of the air-passages on section, and of the amount of space they occupy in proportion to that occupied by the bronchi and air-vesicles proper. It must be understood that these air-passages are really part of the vesicular system of the lung and not of the bronchial system.

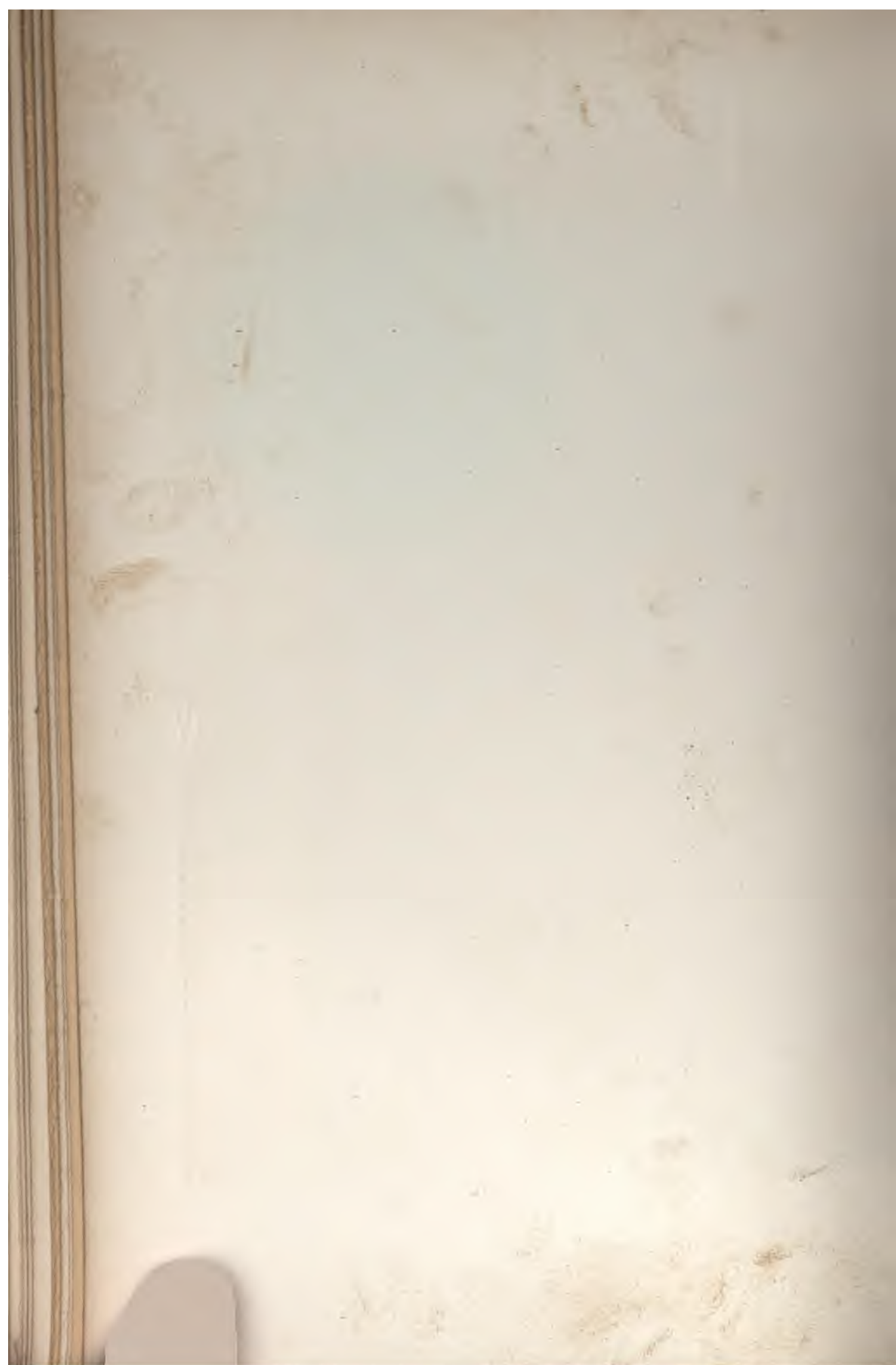
The white or yellow nodules of phthisis seem to be formed in air-vesicles, in single air-passages, or in groups of air-passages, and in and around bronchi.

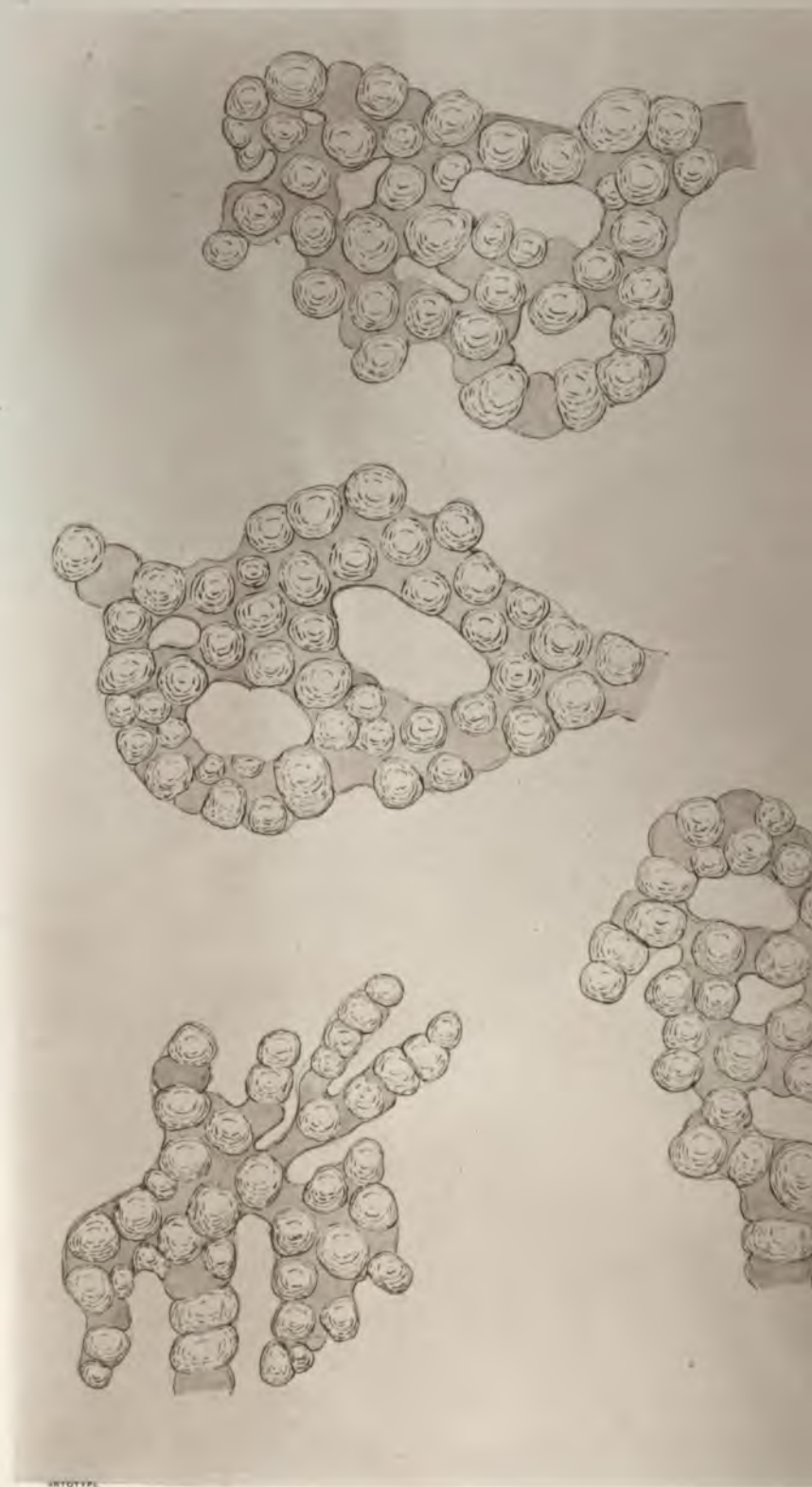
(1.) *The smallest nodules.*—These are not larger than a single air-vesicle or part of an air-passage. In the fresh lung it is not easy to distinguish them from the diffuse hepatization by which they are sur-



ARTOTYPE

HUMAN

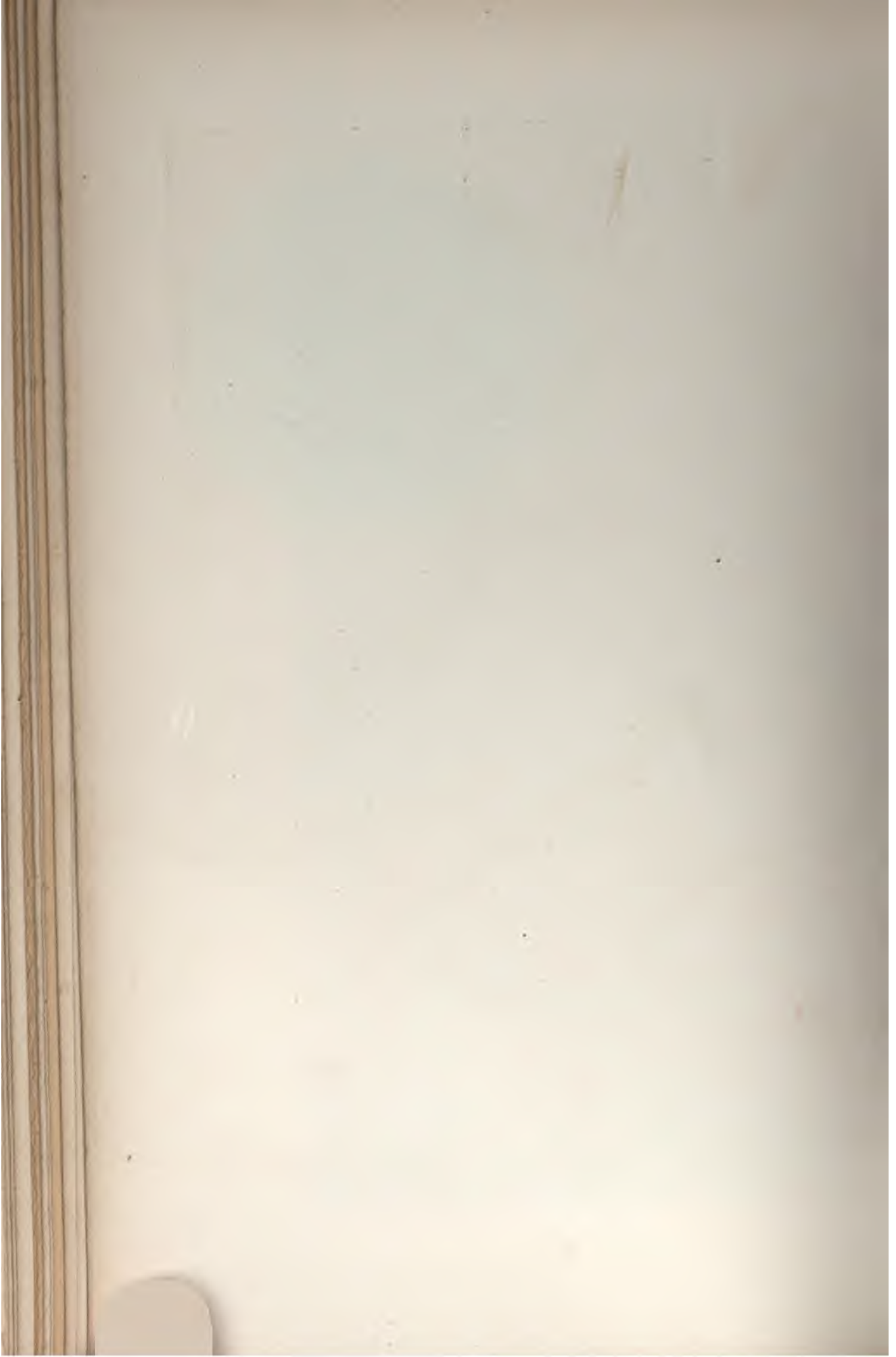


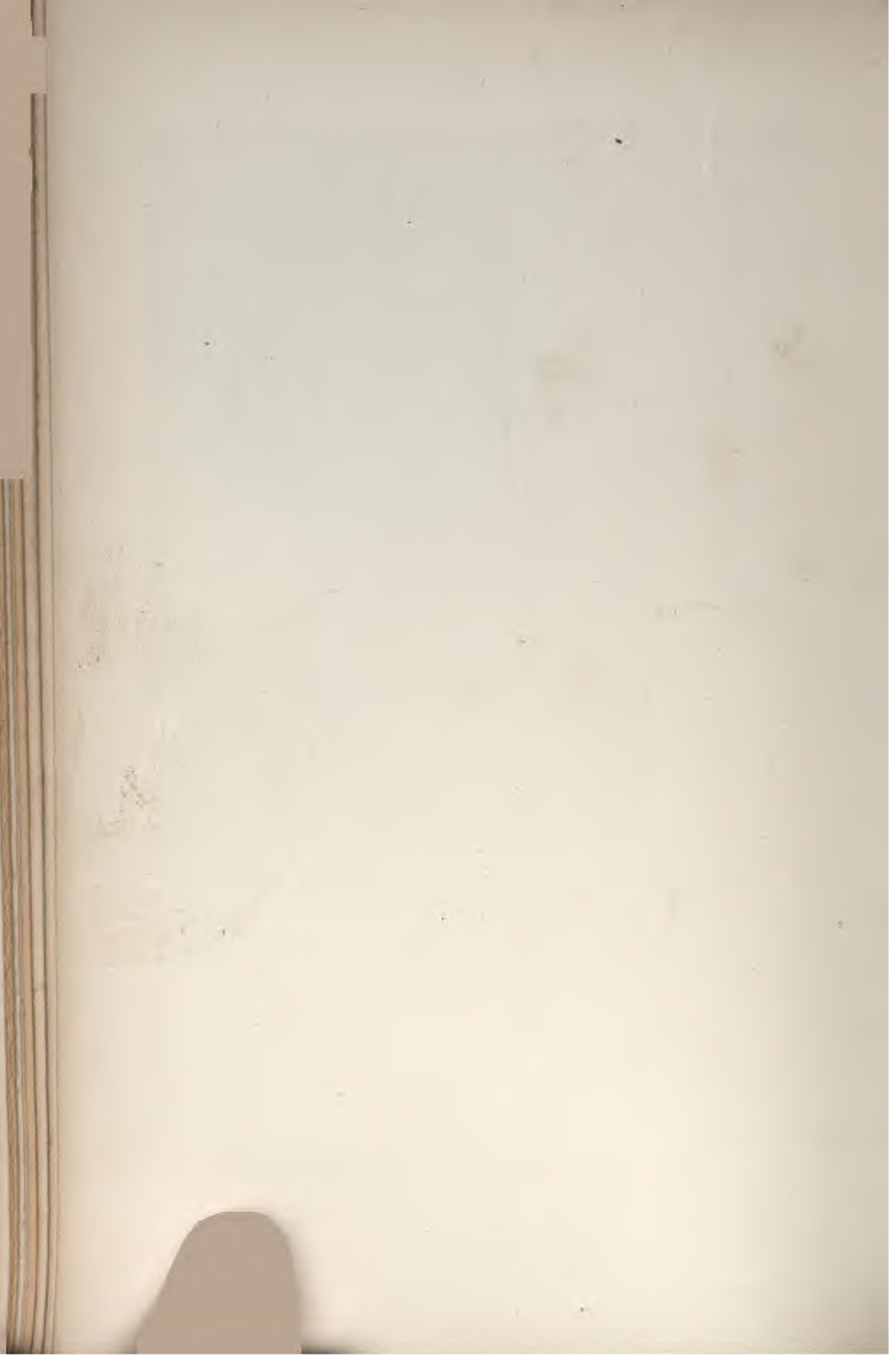


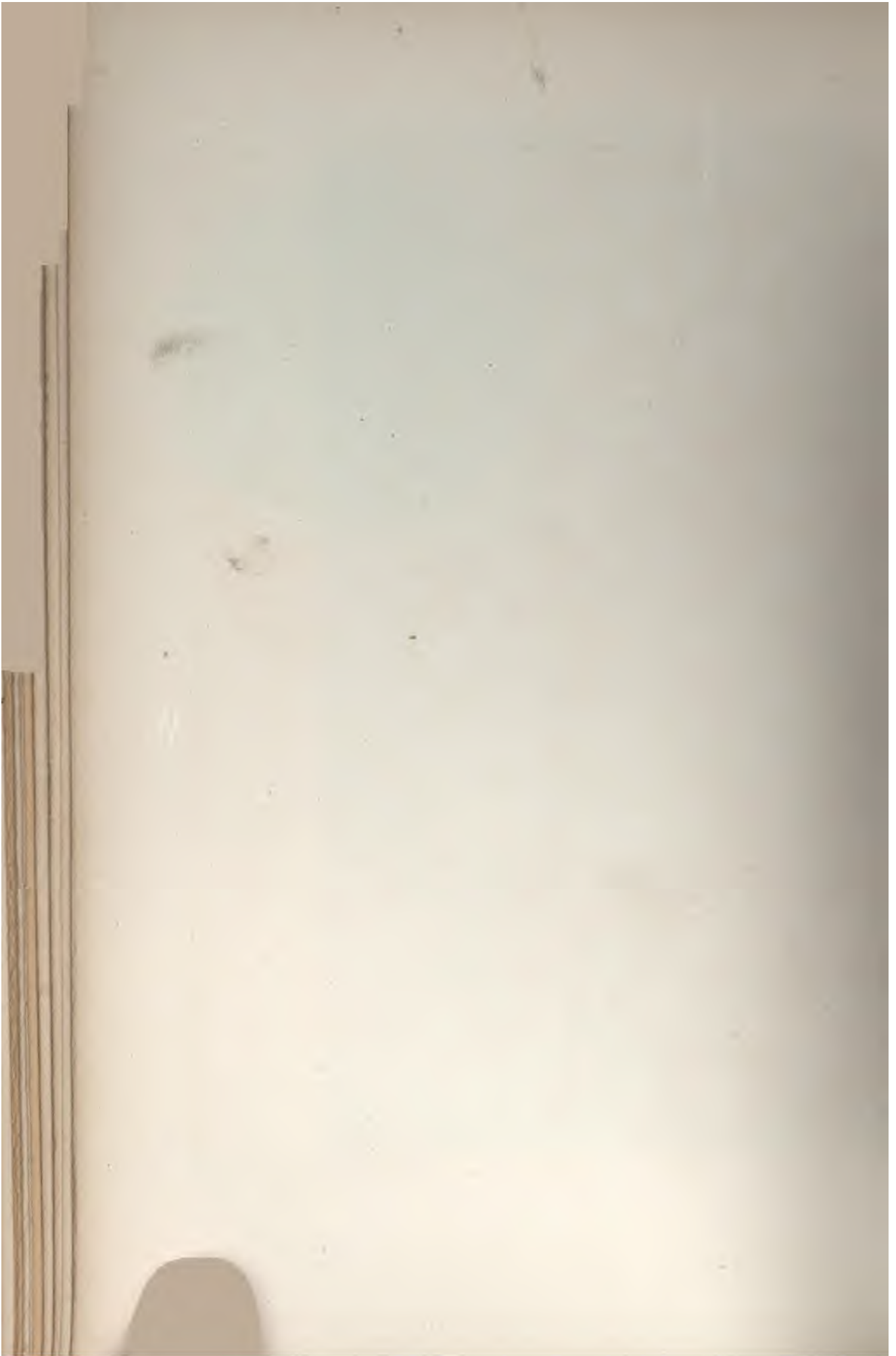
ARTOTYPE

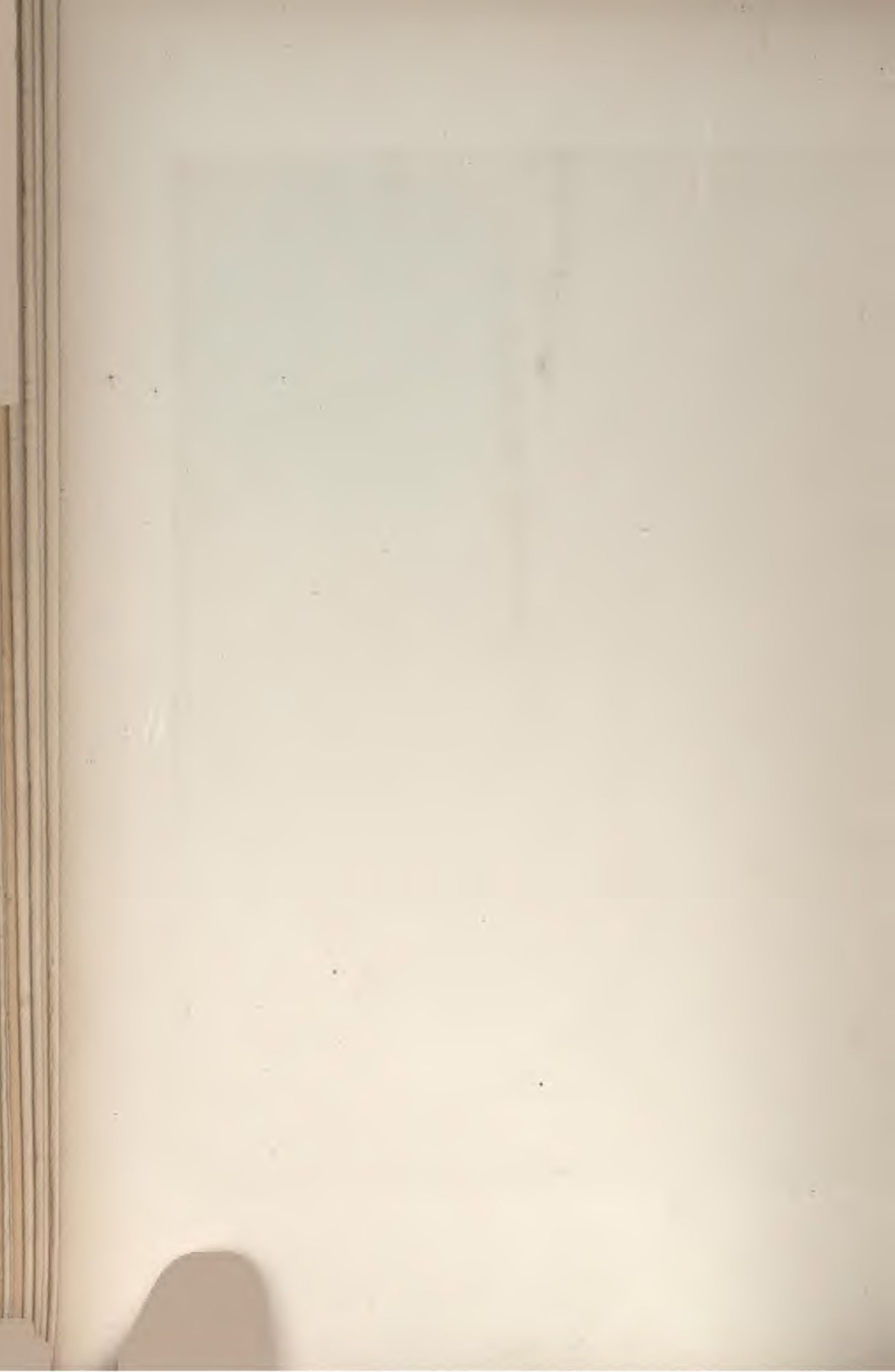
Pla
HUMAN I

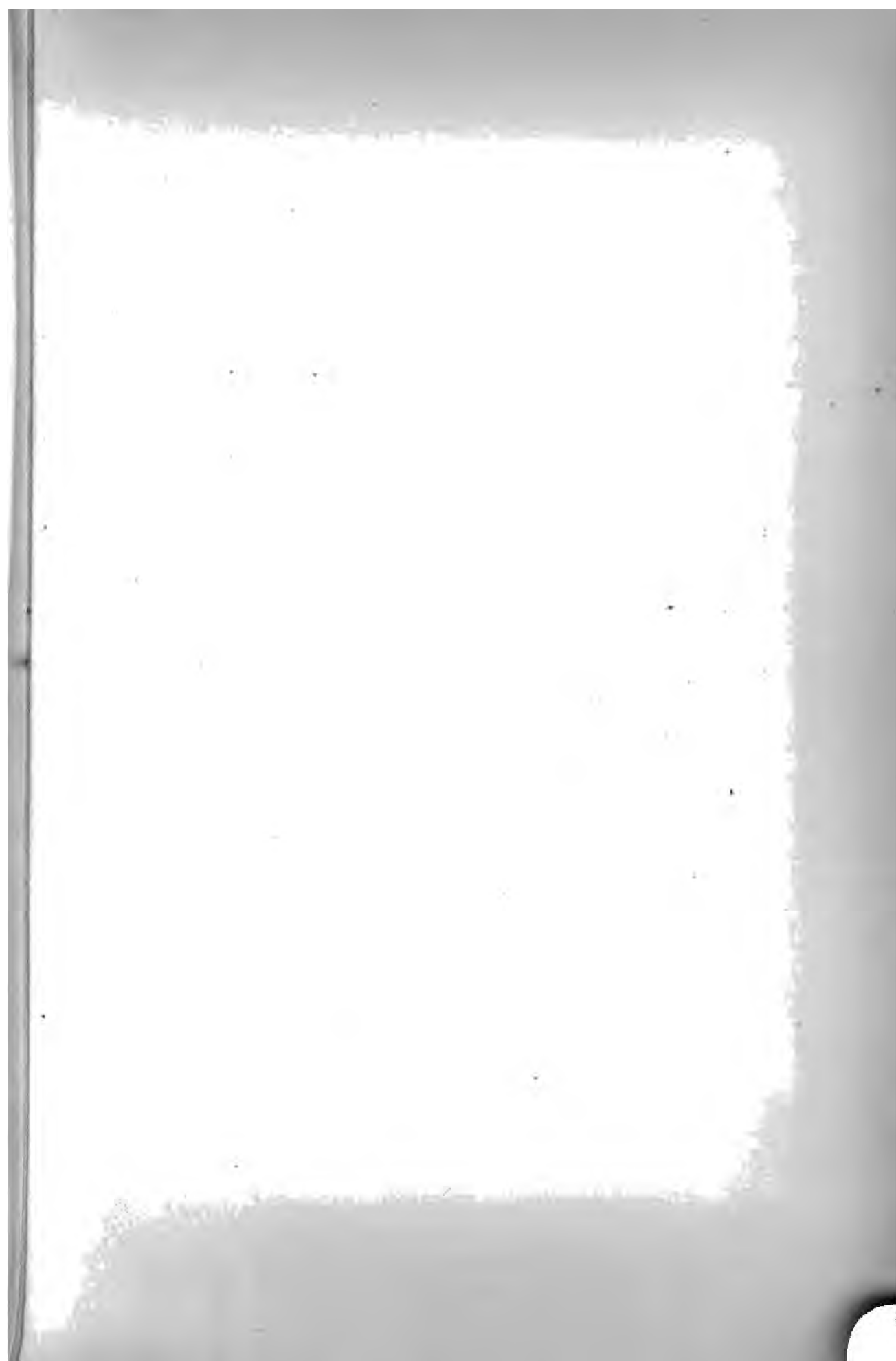














When we examine these peri-bronchitic nodules more closely, it becomes evident that they do not all have the same structure.

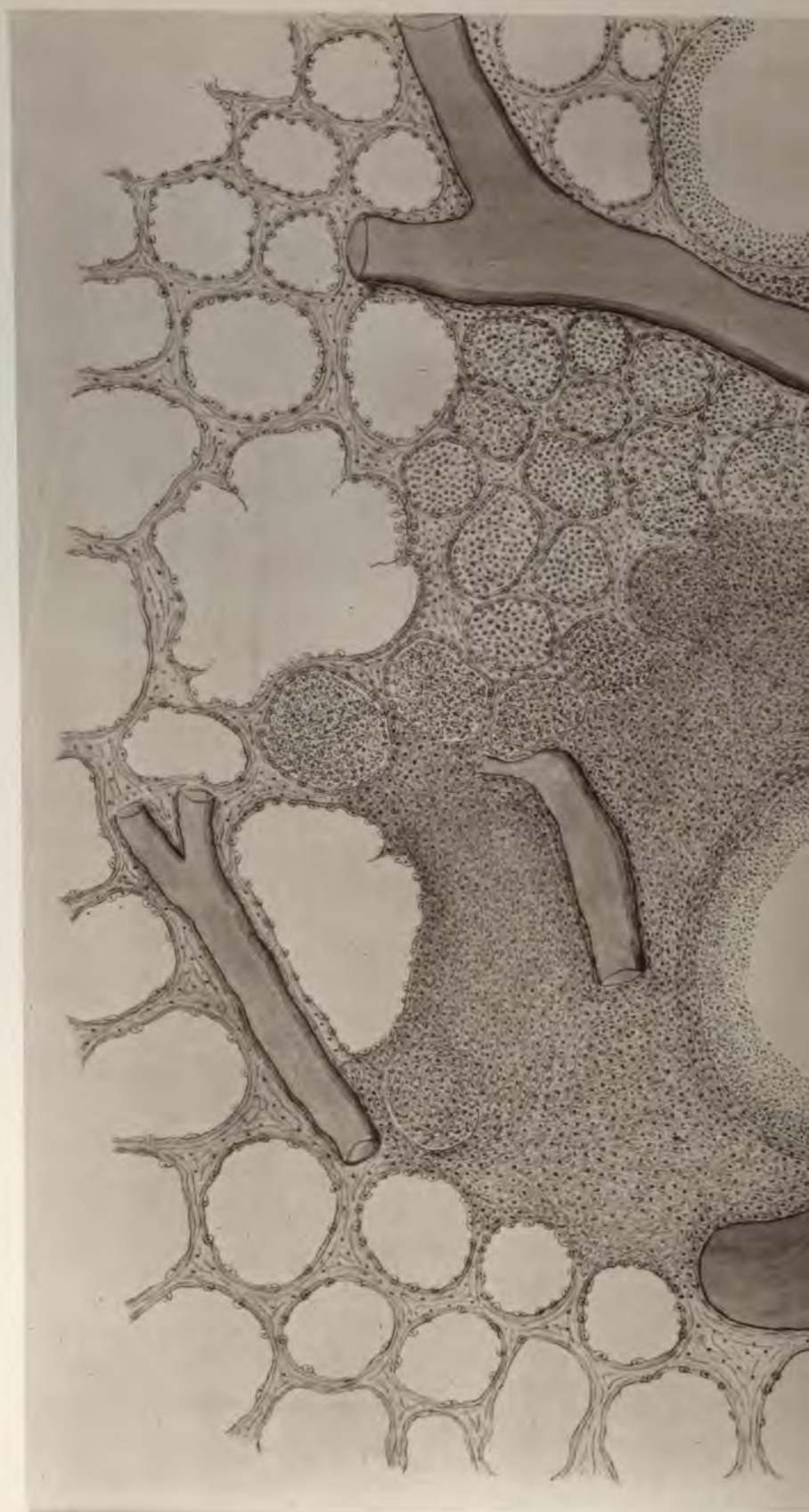
(*a.*) Some of the nodules consist simply of a bronchus filled with pus and epithelium, its walls infiltrated with cells, and of a number of air-vesicles filled with epithelium, fibrine, and pus. Such nodules are identical in their structure with the peri-bronchitic nodules which are formed in the ordinary broncho-pneumonia of children and adults. The advocates of a purely inflammatory phthisis always lay great stress on the character of these nodules.

(*b.*) Other nodules are composed of a bronchus filled with pus and epithelium. The wall of the bronchus is infiltrated with small cells and with tubercle tissue. Around the bronchus is a zone of air-vesicles filled with tubercle, the walls of the vesicles split up and only visible with high magnifying powers. Around this tubercular zone is another outer zone of vesicles filled with epithelium, fibrine, and pus. Plate LXXVI.

(*c.*) Other nodules are composed as before of a bronchus with infiltrated walls, and filled with inflammatory products. Around the bronchus is a continuous zone of tubercle tissue, in which the walls of the vesicles cannot be seen. Artificial injections of the blood-vessels penetrate only very imperfectly into this tubercular zone. Outside of this tubercular zone will be a zone of vesicles filled with epithelium, fibrine, and pus, or with tubercle tissue. Very often the contents of the bronchus, the walls of the bronchus, and even some of the tubercle tissue will undergo cheesy degeneration. Plates LXXVII. and LXXVIII.

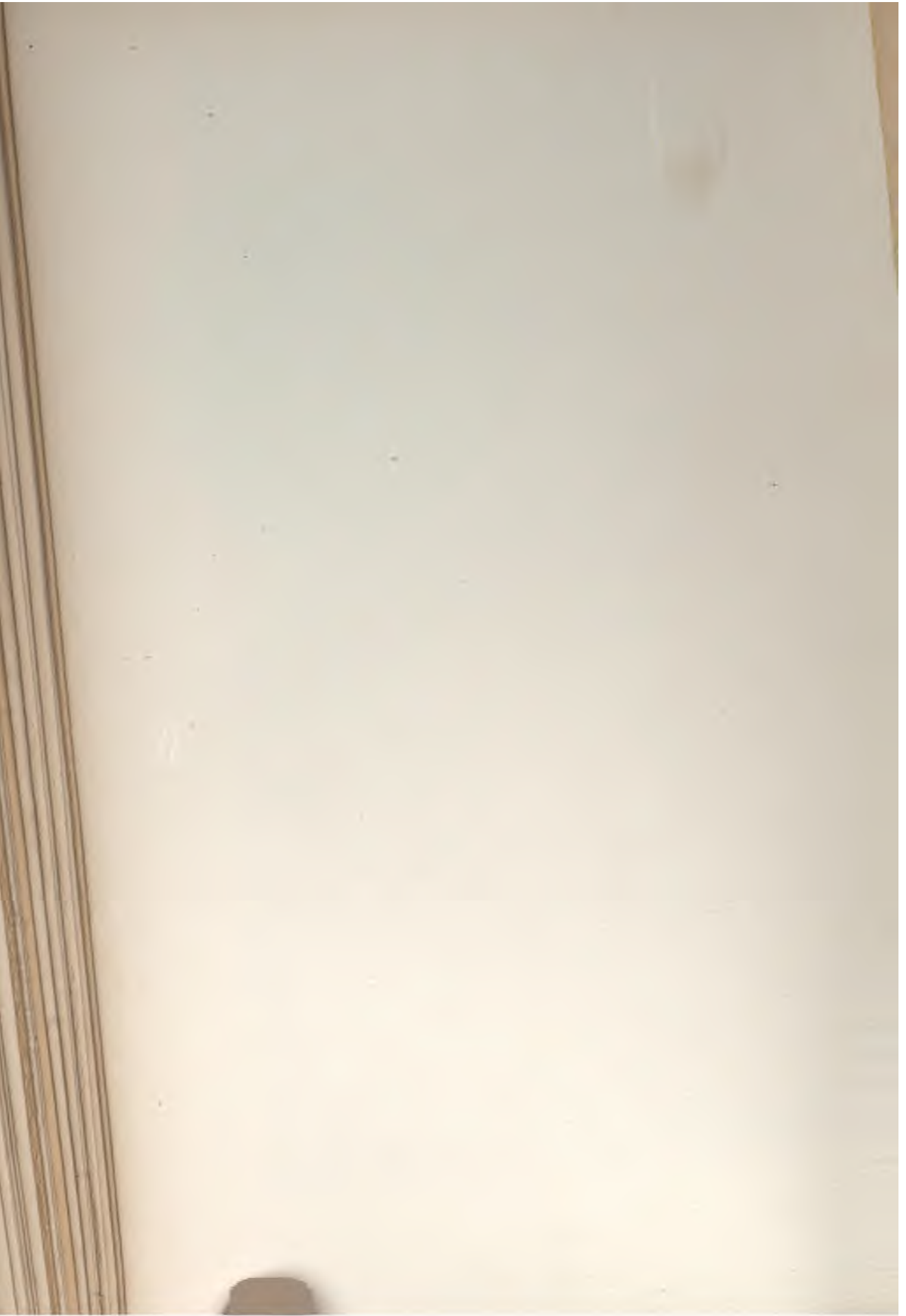
(*d.*) In other peri-bronchitic nodules, although the cavity of the bronchus contains inflammatory products, yet its wall will remain unchanged, and around the bronchus will be air-vesicles filled with tubercle tissue. Such nodules seem to be peri-bronchitic miliary tubercle. Plate LXXIX. It will be observed that in all these peri-bronchitic nodules a large part of the consolidation is due to changes in the air-vesicles.

In connection with peri-bronchitis it is convenient to speak of two lesions which affect the bronchi directly in some cases of acute phthisis.



ARTIST'S COPY

TUBER

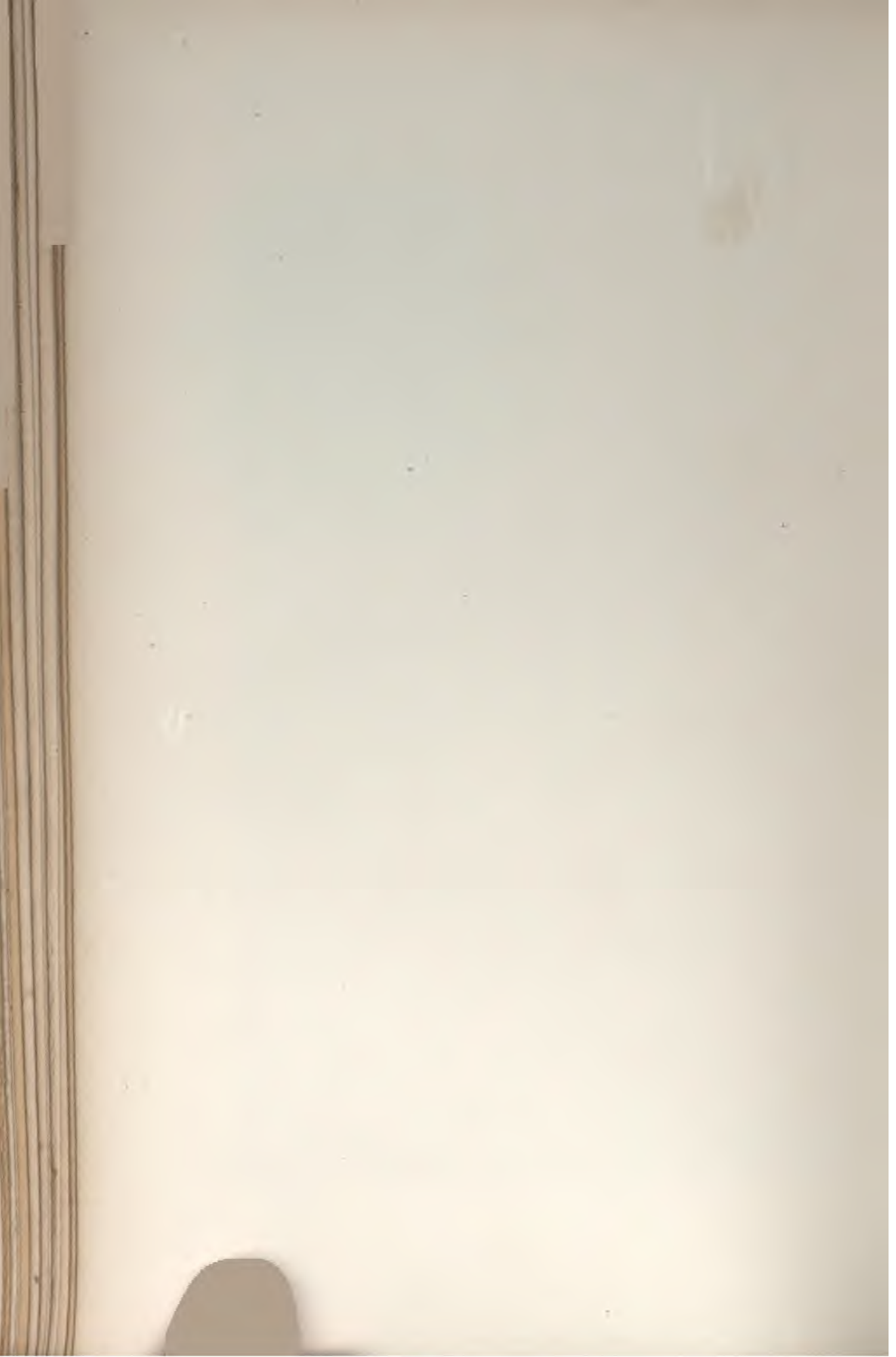


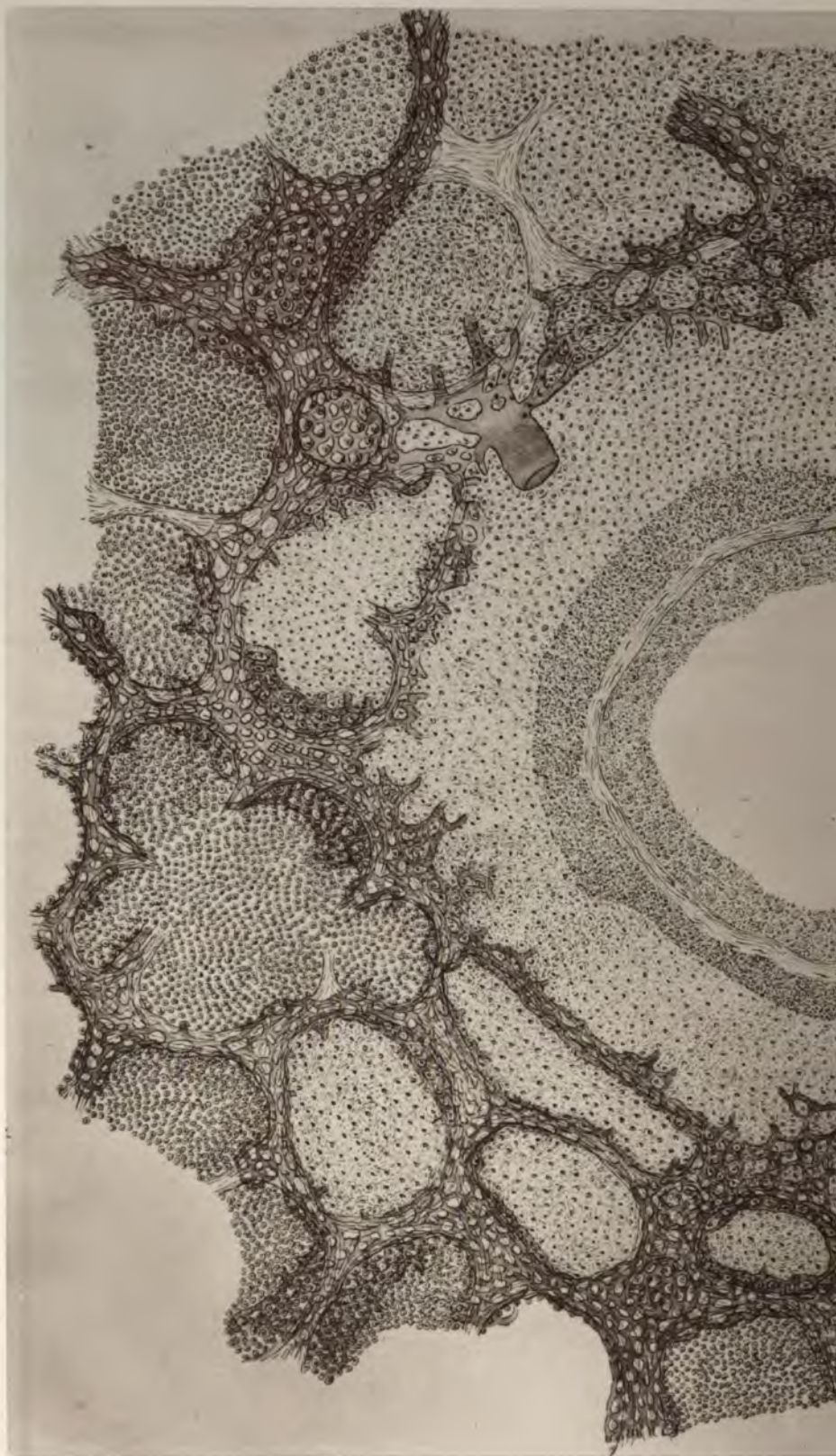




ARTIST'S

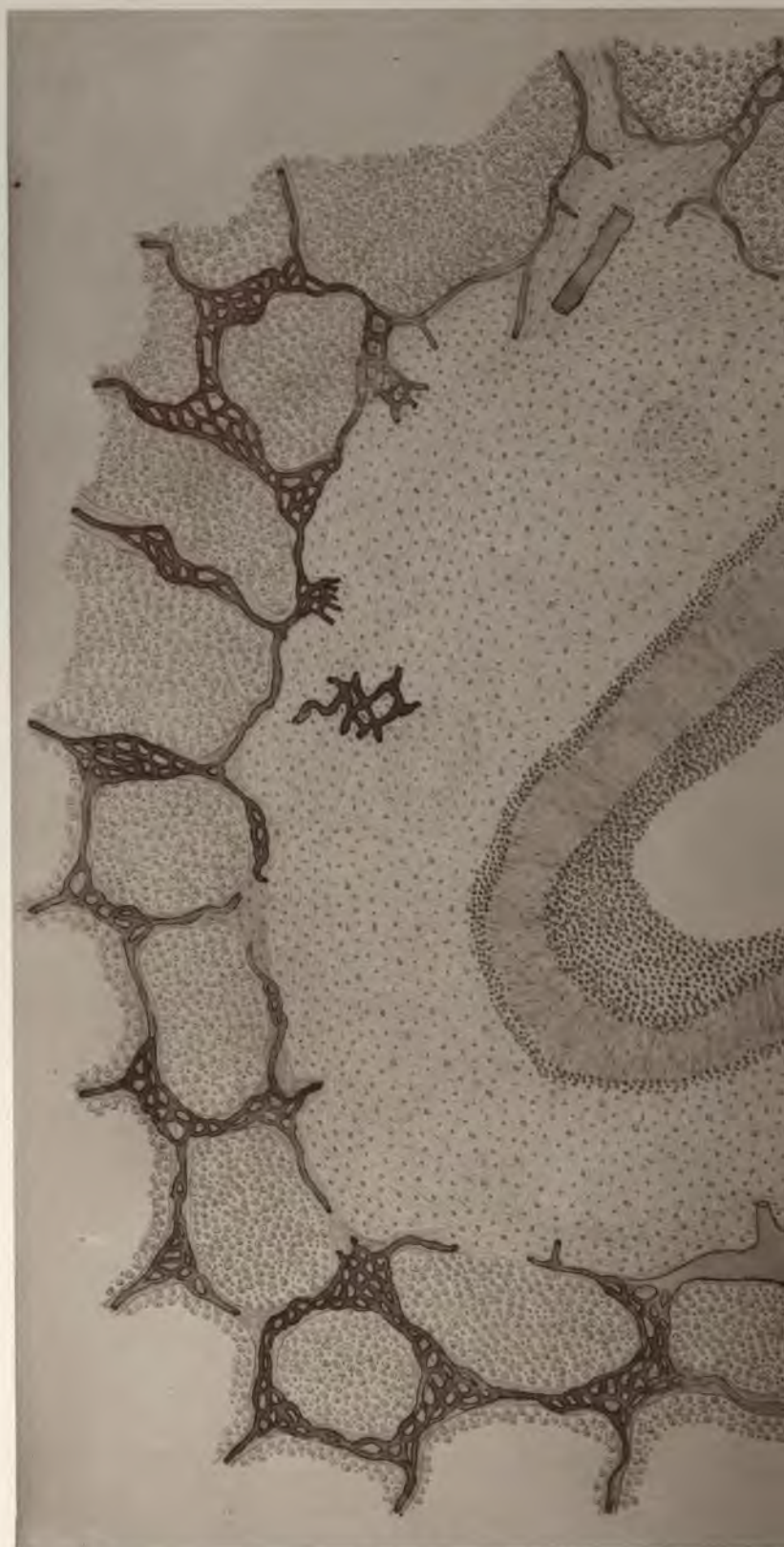
F
TUBERC





ARTOTYPE.

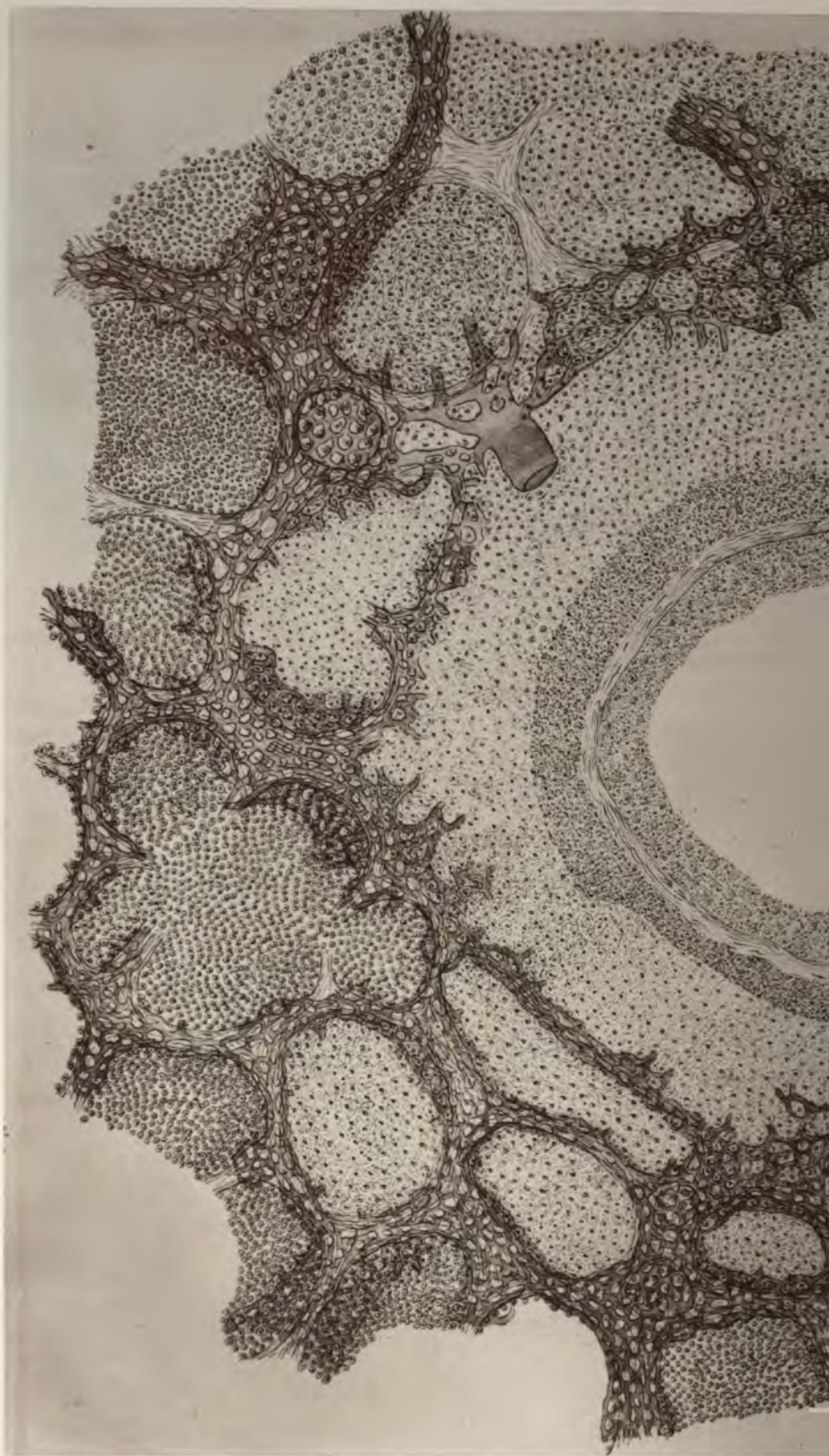
F
TUBER



ARTOTYPE.

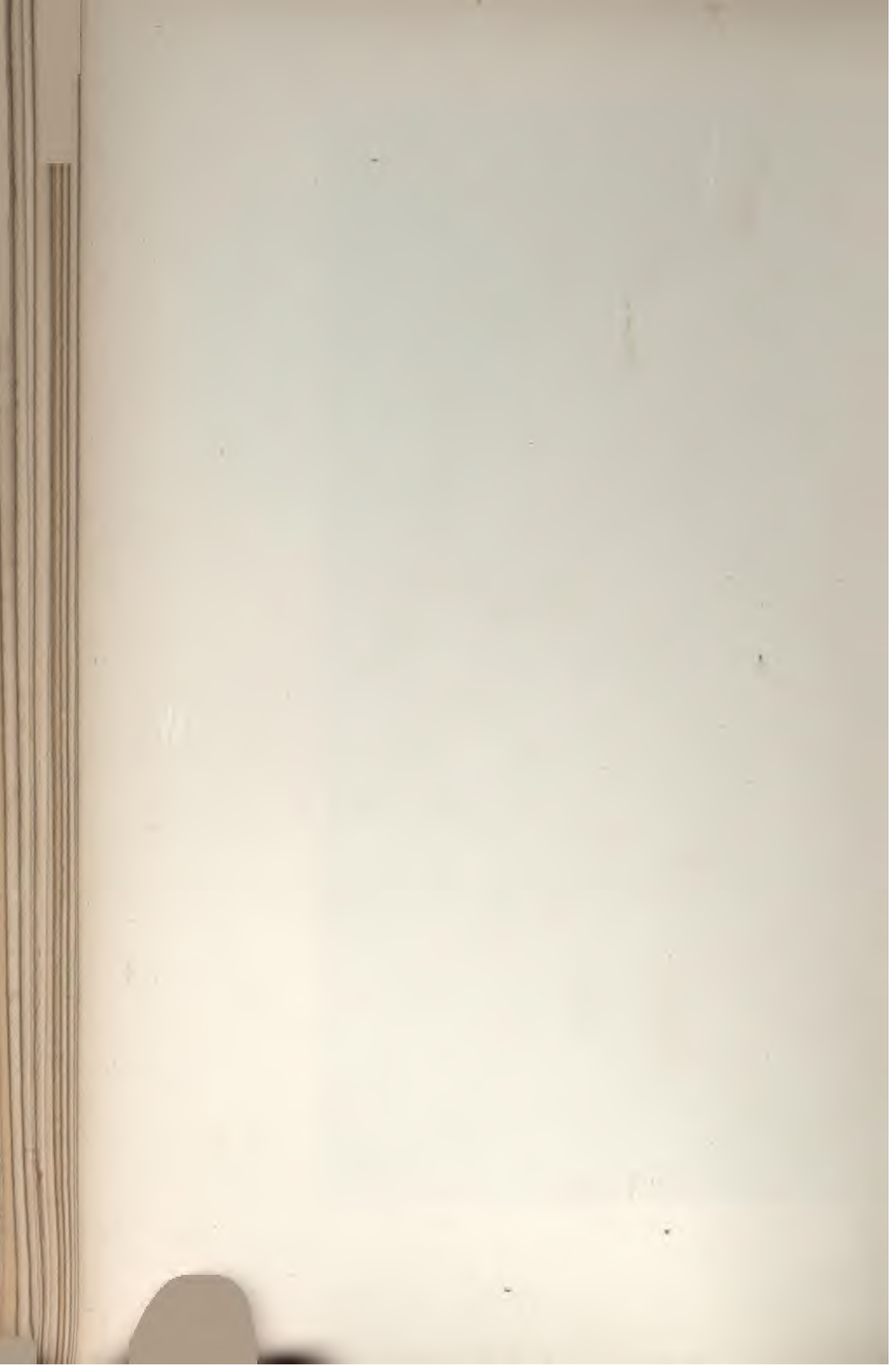
Pl.
TUBERC





ARTOTYPE.

TUBER





HYDRA

P
TUBERCI







ARTOTYPE.

S. WERNSTADT N. 7

Plate LXXXI. x 8

ACUTE PHTHISIS.



1. The walls of some of the larger bronchi are infiltrated with tubercle tissue which undergoes cheesy degeneration. The cavity of the bronchus is dilated, and contains inflammatory products also in a condition of cheesy degeneration. The adjacent air-vesicles may be unchanged, or may contain tubercle tissue, or pus, fibrine, and epithelium. This change affects especially the larger and medium-sized bronchi, sometimes a large bronchus and its branches. The dilatation may be sufficient to form a cavity of some size, which may be further enlarged by the disintegration of the cheesy bronchial wall. Plate LXXX. For the most part in these bronchi the cheesy degeneration of the wall is so advanced that no structure can be made out, but sometimes one can find a place which seems to show the previous infiltration of tubercle tissue.

2. There may be a general dilatation of most of the bronchi in a considerable portion of the lung without any marked change in their walls, and with only a moderate amount of inflammatory products in their cavities. This change is especially apt to affect the medium-sized and small bronchi. The lung tissue between the dilated bronchi is usually consolidated. When such a lung is cut it looks as if it were honey-combed with small cavities, but these cavities are only sections of the dilated bronchi. Plate LXXXI.

The larger nodules found in acute phthisis seem to be of three kinds.

1. Peri-bronchitic nodules, such as have been already described.

2. Bodies resembling miliary tubercles in their structure, but much larger—the “tubercules massifs” of French writers. These nodules have just the same structure as many of the ordinary miliary tubercles already described, but they are on a larger scale, and the centre is regularly the seat of cheesy degeneration.

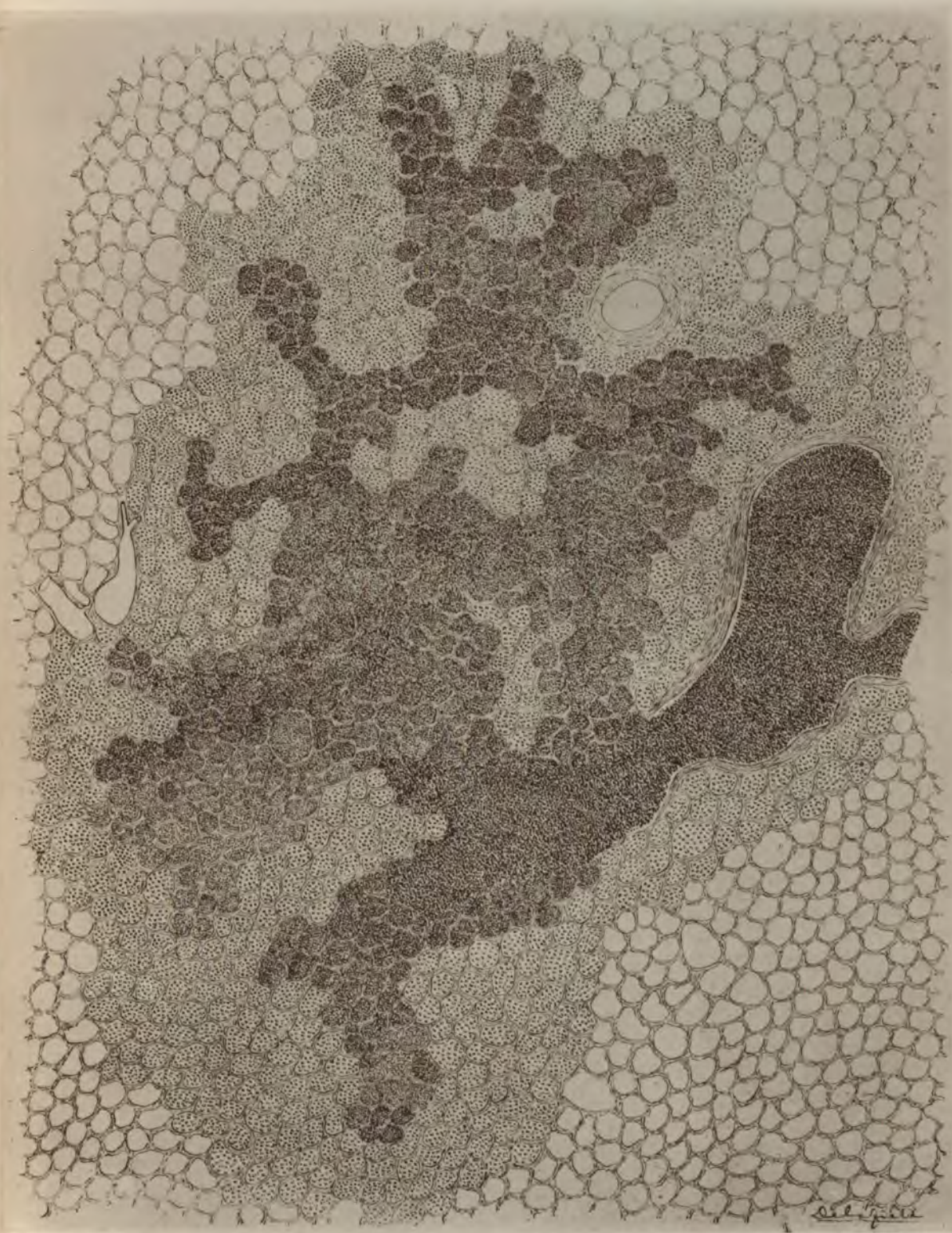
3. Nodules formed by a number of air-vesicles in the condition of coagulation necrosis.

It is necessary here to have precise notions as to what is meant by the terms “cheesy degeneration” and “coagulation necrosis.”

Cheesy degeneration, or metamorphosis, is a term made popular by Virchow. “It is,” he says, “the second stage, or the necrobiotic result

of an originally hyperplastic growth-process, and the cheesy substance is nothing but the dead remains of broken down tissue. In this necrobiosis both the newly formed and the old parts die; the circulation stops, and the vessels disappear; the cells degenerate partly by an incomplete fatty metamorphosis, partly by a shrinkage from the loss of water. Thus is formed the totally anæmic, dry, dense, and almost amorphous mass." Such a change may take place in inflammatory products or in new growths. The term then expresses a perfectly well-defined condition, namely, the degenerative changes which take place in inflammatory products, new growths, and old tissues, and result in the transformation of these different elements into an amorphous mass composed of granular matter, fat, and shrivelled tissue. The process is one of death, disintegration, fatty degeneration and desiccation.

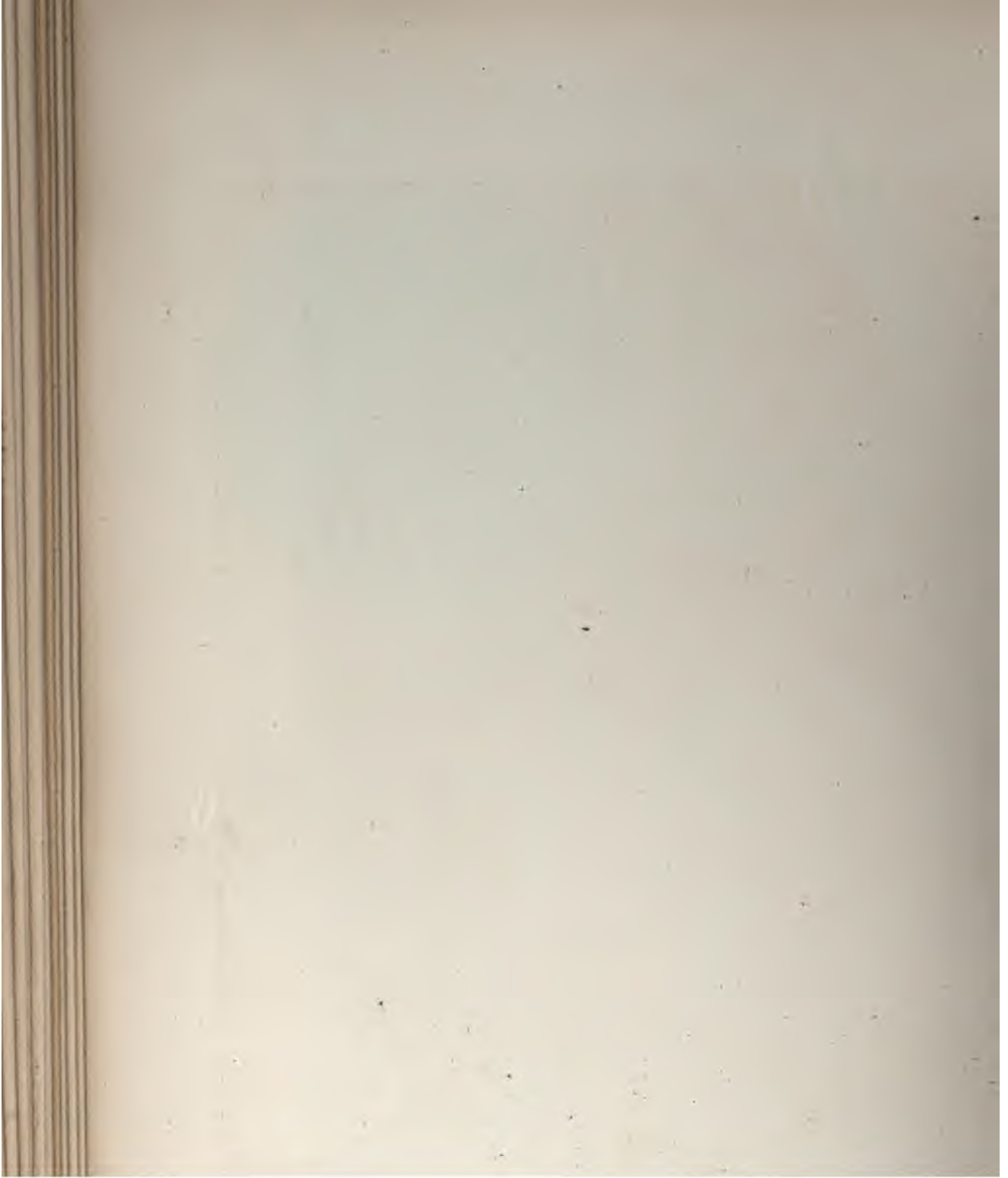
Coagulation necrosis is a term introduced by Cohnheim to designate a condition to which attention has been called by Weigert. It is found—if the vascular supply is shut off from a portion of tissue, which remains surrounded by other tissue in which the blood circulates—that this anæmic portion of tissue undergoes certain changes, which are of the nature of a coagulation. This is best seen in the kidneys and spleen. If, for example, in the spleen one of the arterial branches is stopped by an embolus, a corresponding portion of the spleen will become anæmic, and will appear as a white, wedge-shaped mass, sharply defined from the surrounding red normal spleen. If such a white infarction has existed but a short time, there is hardly any difference between the appearance of its anatomical elements and those of the surrounding spleen, except that it will be differently affected by staining fluids. If the infarction is older, then the cells become smaller, the nuclei disappear, the cell-bodies assume a peculiar shining appearance. In this condition the infarction may remain for a long time, but in the meanwhile changes take place at its periphery. There is a new growth of connective tissue so that the infarction may be surrounded by a zone of new connective tissue. Finally, cheesy degeneration may set in at the centre of the infarction, and extend so as to involve the greater part of it.

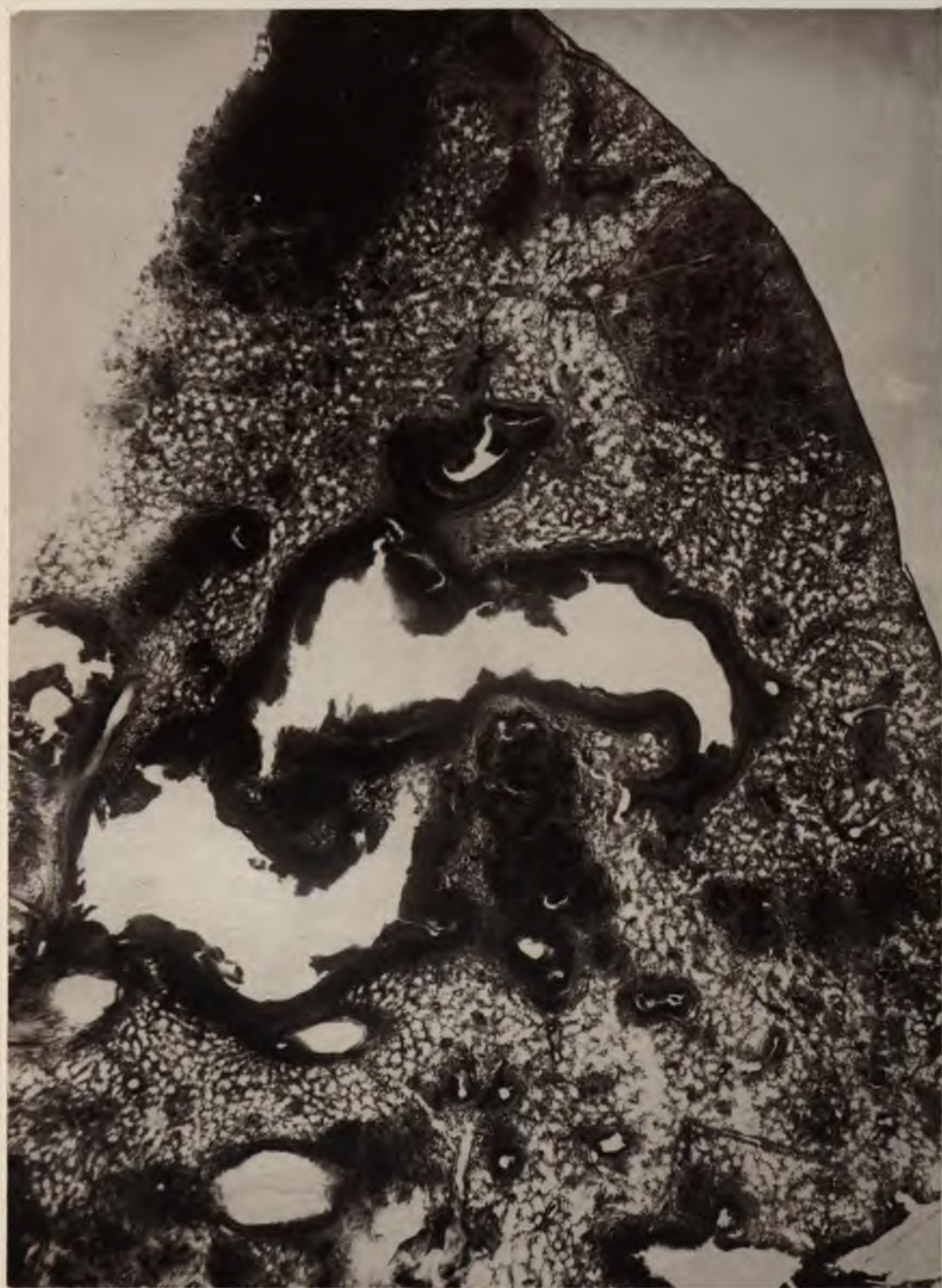


ARTOTYPE

E. HENSTADT N. Y.

Plate LXXXII. x40
ACUTE PHTHISIS.



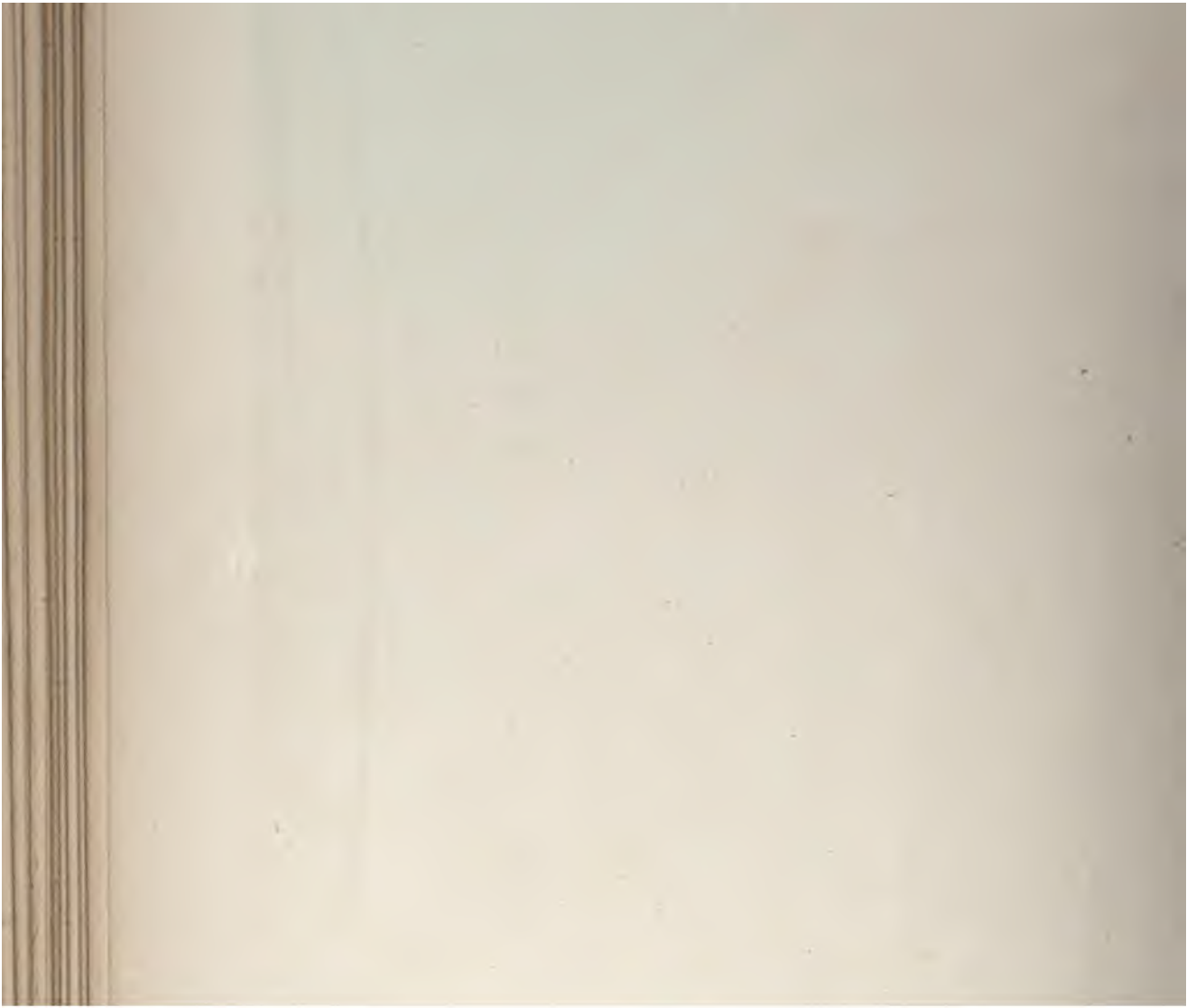


ARTIST TYPE

C. WERSTADT N. Y.

Plate LXXX, x 5

ACUTE PHTHISIS.





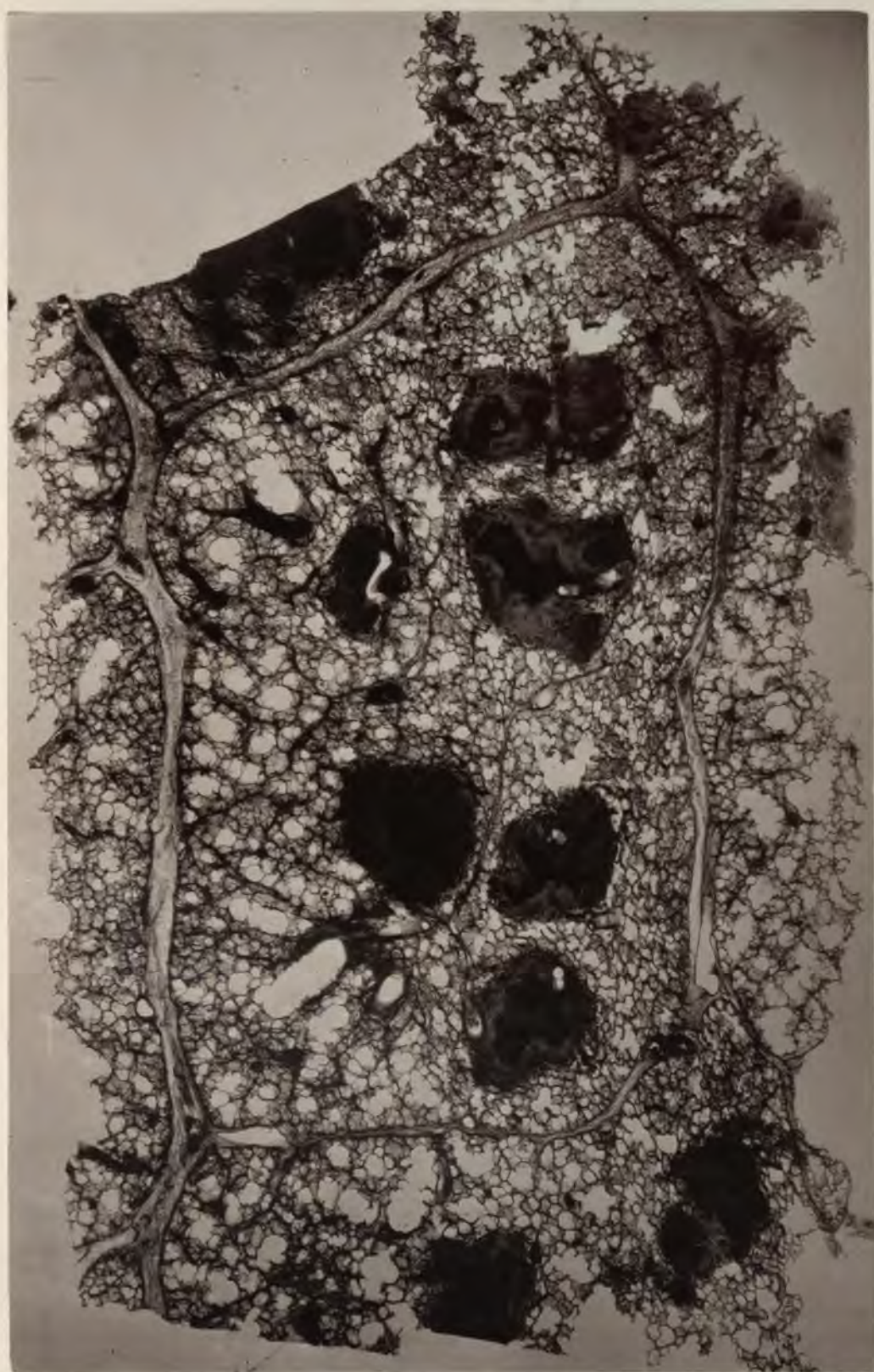
ARTOTYPE.

T. BERSTADT N. Y.

Plate LXXXI. x 8

ACUTE PHTHISIS.





ARTOTYPE

E. HERSTADT N. Y.

Plate LXXXV. x 11

ACUTE PHTHISIS.



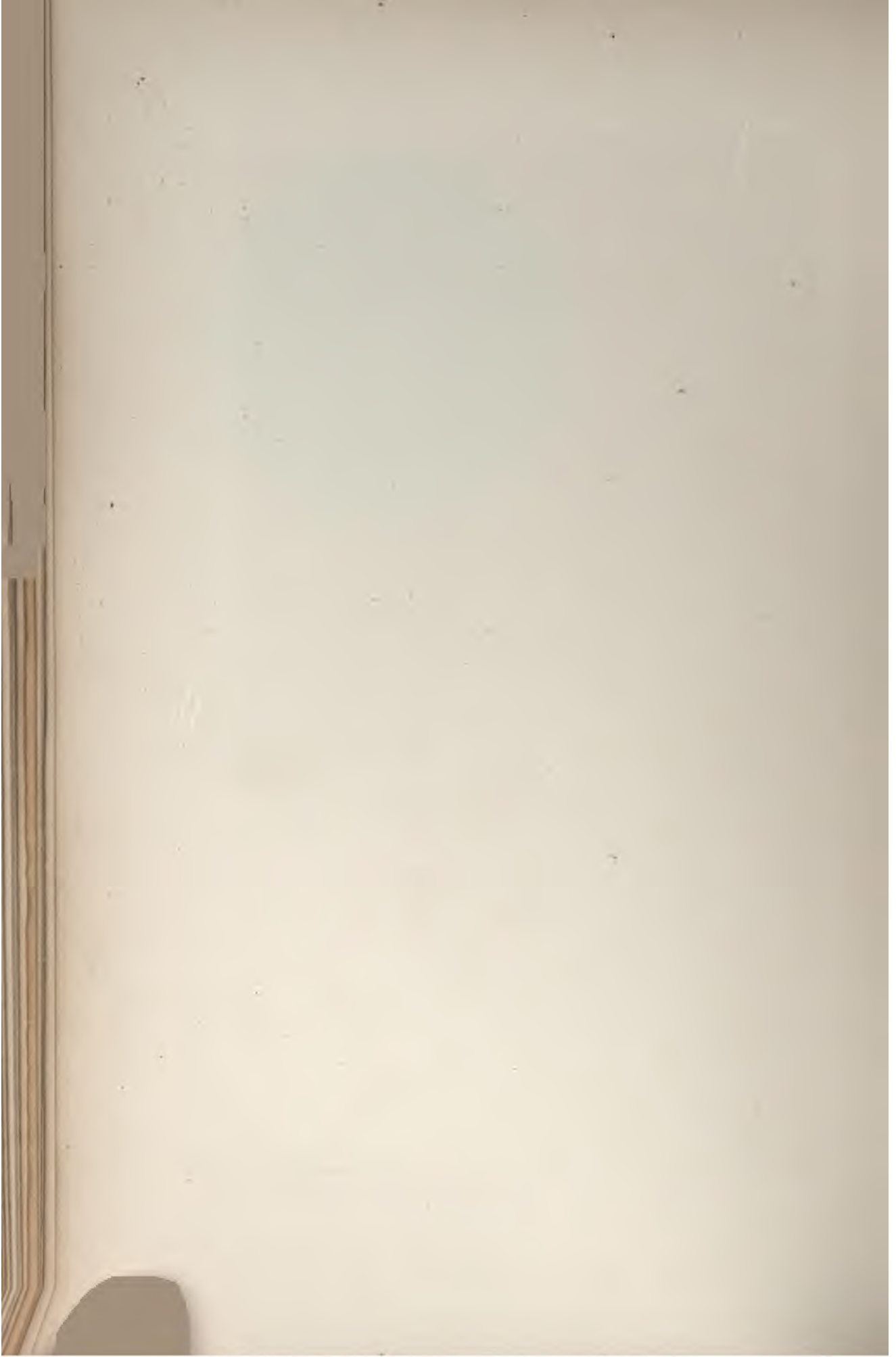


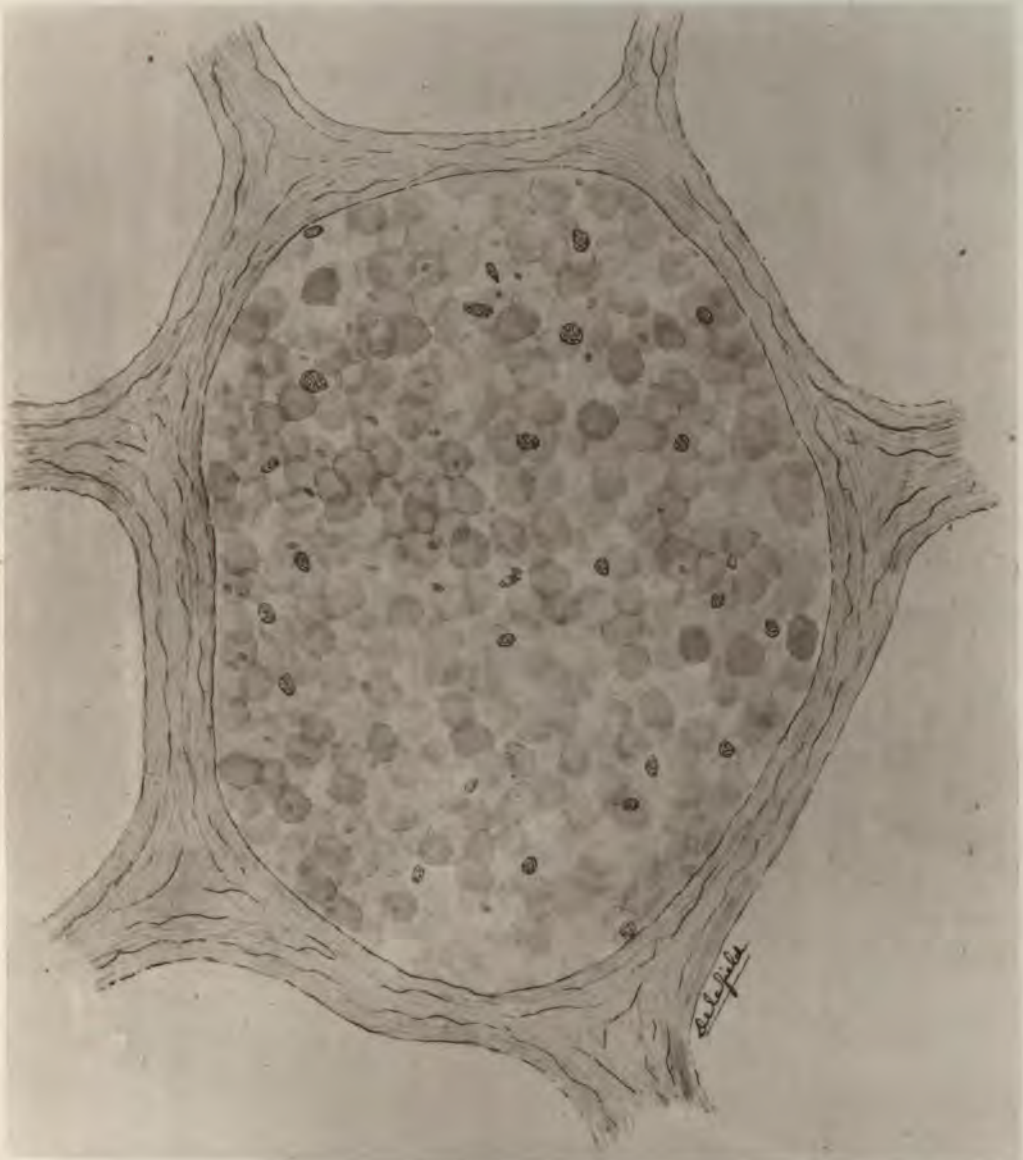
ARTOTYPE

HERBSTADT & S.

Plate LXXV. x 4

ACUTE PHTHISIS.





ARTOTYPE.

E. HERSTADT N. Y.

Plate LXXXVI. x850
ACUTE PHTHISIS.

Cheesy degeneration and coagulation necrosis are, therefore, totally different conditions. But tissues which are first the seat of coagulation necrosis may afterward undergo cheesy degeneration. Although the two processes are so distinct, yet the gross appearances produced by them may be very similar. Portions of tissue, the seat of coagulation necrosis may resemble very closely portions of tissue which have undergone cheesy degeneration. It is in the study of phthisis particularly, that this similarity has led to confusion.

In acute phthisis the nodules due to such a process of coagulation necrosis are regularly present and form a very important part of the lesion.

The simplest form of such a coagulation necrosis nodule is seen in Plate LXXXII. Here is a portion of a small bronchus and a single air-passage in the condition of coagulation necrosis surrounded by a zone of ordinary hepatization. Plate LXXXIII. shows the same condition more extensively developed. Here the areas of coagulation necrosis are so large and so close together as to form a nearly continuous hepatization.

A more complex form of nodule is represented in Plates LXXXIV., LXXXV., and LXXV. Here are nodules with dark centres and light peripheries, the larger nodules being made up of several smaller ones. In these nodules the dark centres are areas of coagulation necrosis, the light peripheries are made up of tubercle tissue.

These are the two forms of coagulation necrosis nodules. In each form there is a centre of air-vesicles filled with inflammatory products in a condition of coagulation necrosis; but around such a centre there may be merely a zone of ordinary hepatization, or a zone of tubercle tissue.

If we look more closely at these areas of coagulation necrosis we see that their vascular supply is entirely cut off, and that artificial injections do not penetrate them. The outlines of the walls of the vesicles are visible, but the walls are thin, transparent, recognizable principally by their elastic fibres. The cavities are filled with polygonal cells and shrivelled nuclei. The cells are pale, shining, devoid of nuclei, and have the characteristic appearance of cells in the state of coagulation necrosis. Plate LXXXVI. In some vesicles, however,

there is nothing but coagulated fibrine. These vesicles filled with fibrine are not as opaque as the others, so that a mottled effect is produced, as seen in Plate LXXXIII.

There is nothing especial to be noticed in the zone of ordinary hepatization which surrounds the necrotic centres. The cavities of the vesicles are filled with epithelium, pus, fibrine, and granular matter in varying proportions. The walls of the vesicles are unchanged, their vessels are pervious. It is to be noticed that there is no appearance of a gradual transition between the necrotic centres and the hepatized peripheries. There are no vesicles giving pictures of coagulation necrosis in different stages. All the necrotic portion is in the same condition, and is separated abruptly from the surrounding hepatization.

In those nodules which have a peripheric zone of tubercle tissue, we get, with a higher magnifying power, such a picture as is seen in Plate LXXXVII., which represents a section through a small nodule. The necrotic centre is sharply separated from the tubercular zone, and this tubercular zone gives an indistinct appearance of being made up of altered air-vesicles.

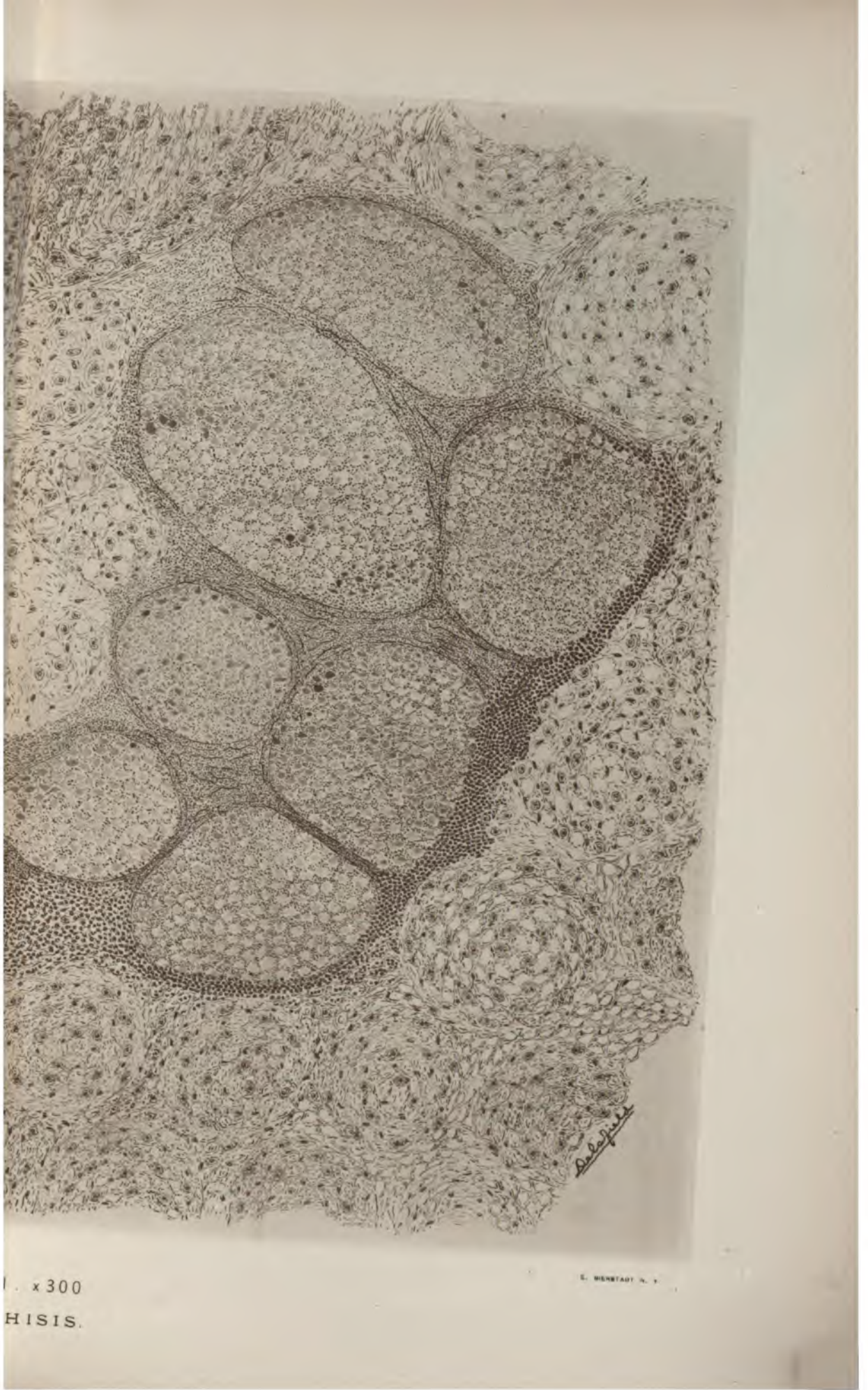
If we examine this tubercular zone more closely, we find that there are changes in the walls of the air-vesicles and in their cavities. The walls are changed into tubercle tissue. At first the blood-vessels in the walls remain pervious, but as the transformation of the walls becomes more complete, the vessels disappear. The cavities of the vesicles are partly or completely filled with tubercle tissue, epithelium and pus. The tubercle tissue grows inward from the walls of the vesicles, or is formed in their cavities; it has the regular structure of a delicate basement-substance, more or less reticulated, provided with nuclei, and enclosing spaces containing large polygonal nucleated cells. Giant cells are sometimes present, but are not constant. Plates LXXXVIII., LXXXIX., XC. The process seems to be of the same character as the formation of diffuse tubercle tissue already described in connection with chronic miliary tuberculosis.

These nodules may remain in the condition just described up to the time of the patient's death. But in some cases they undergo further changes. Cheesy degeneration begins at the centre of the necrotic



ARTOTYPE.

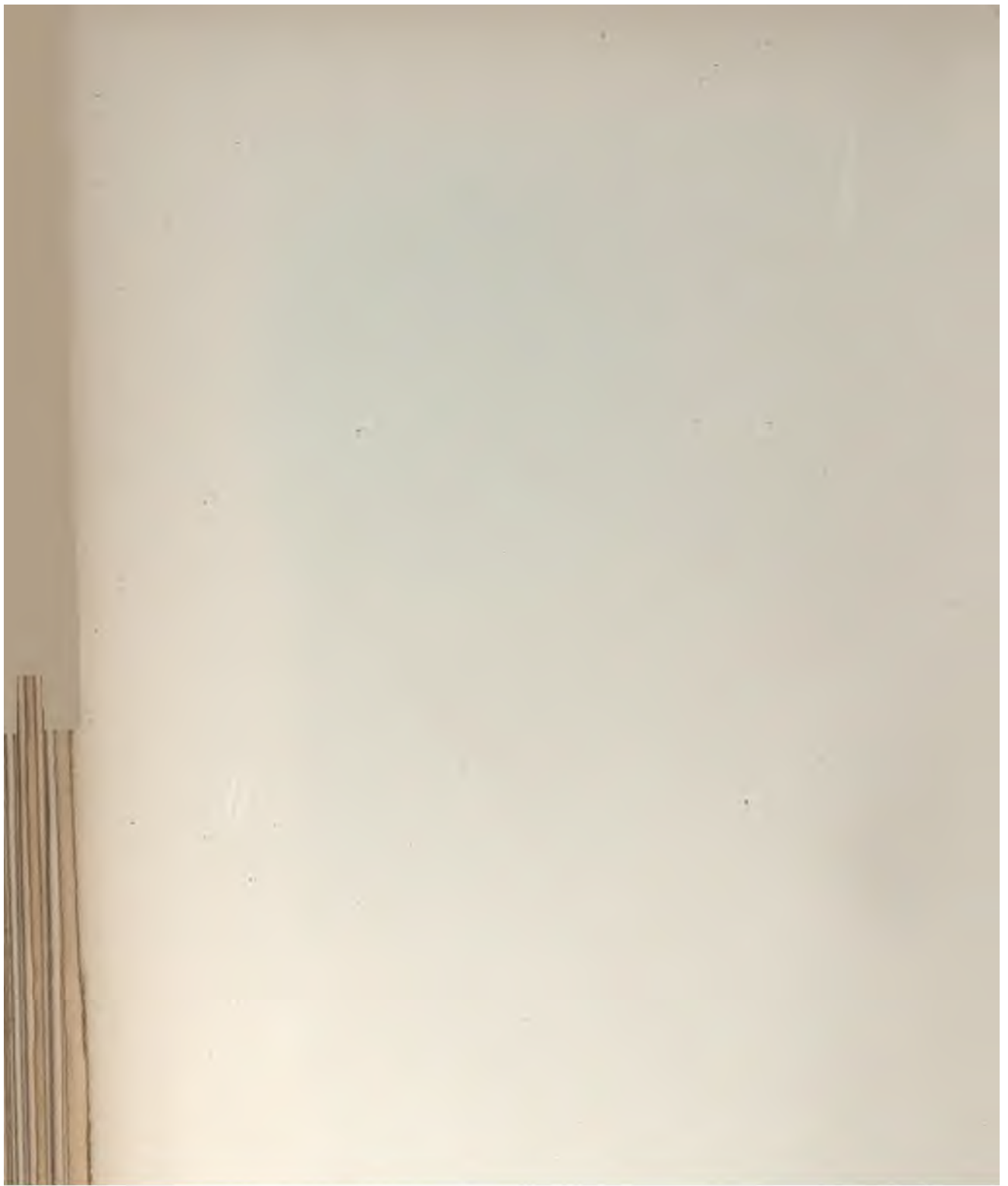
Plate LXXXVI
ACUTE PHT

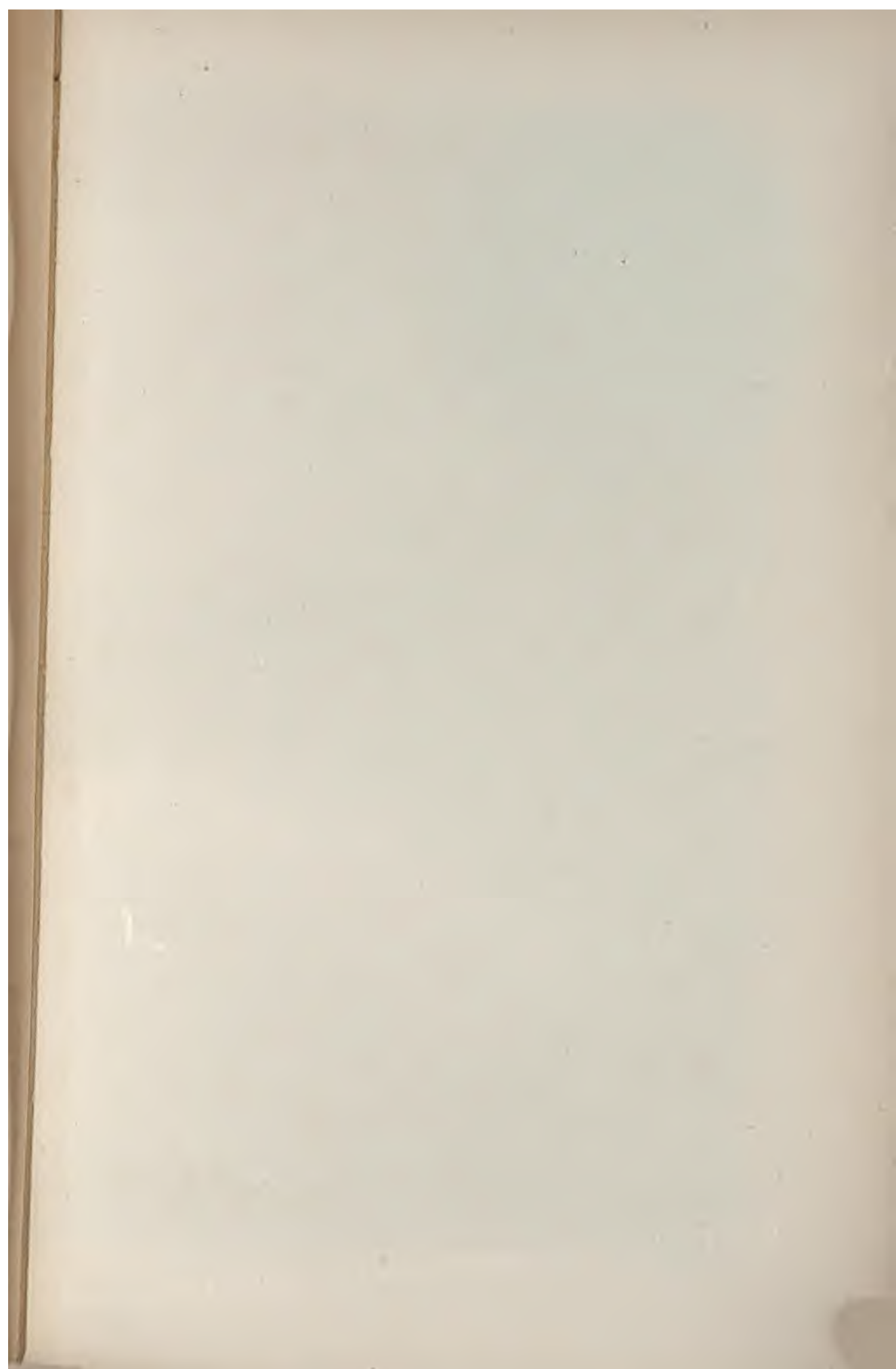


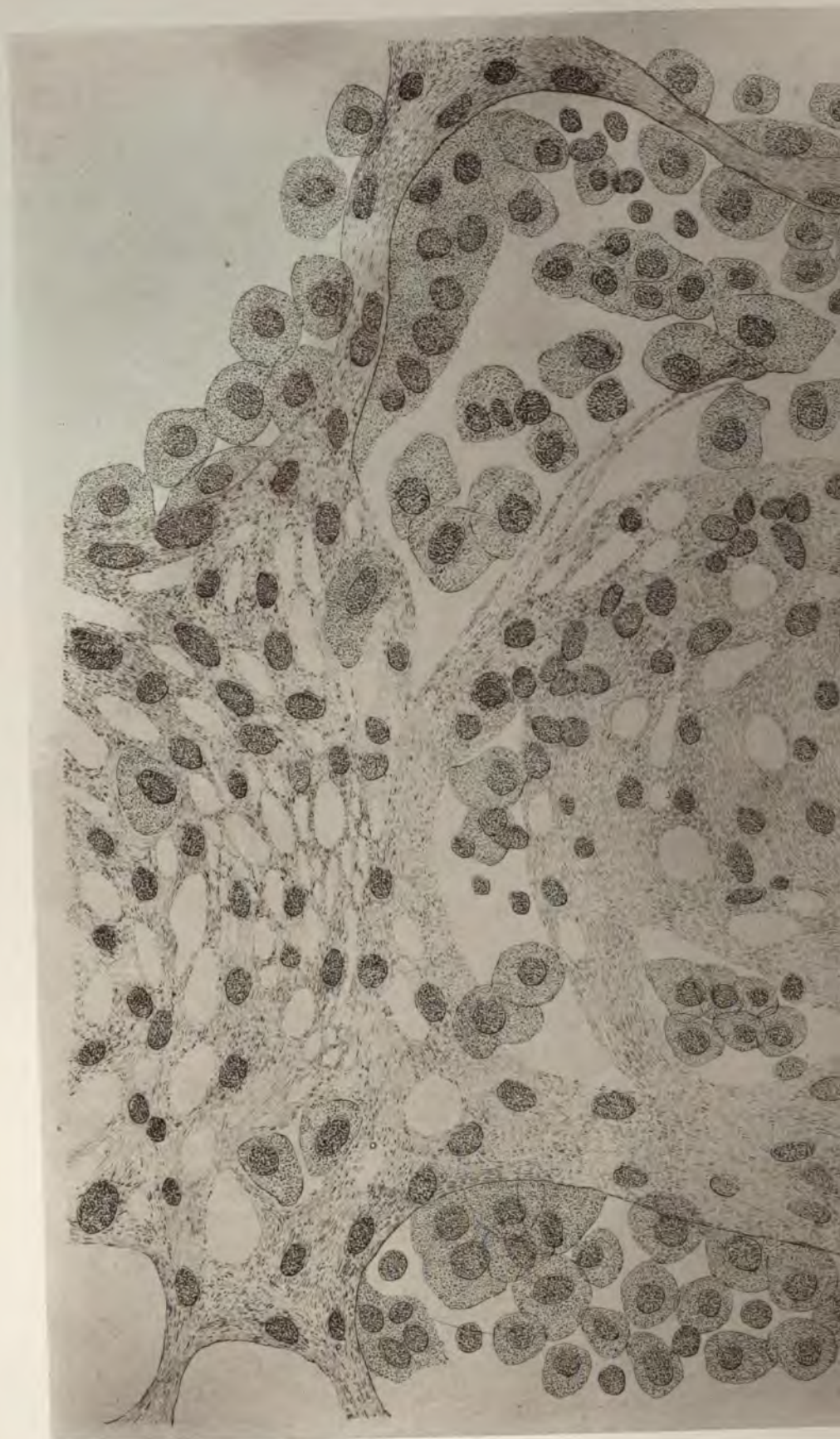
x 300

HISIS.

E. WENSTADT N. Y.







ARTOTYPE.

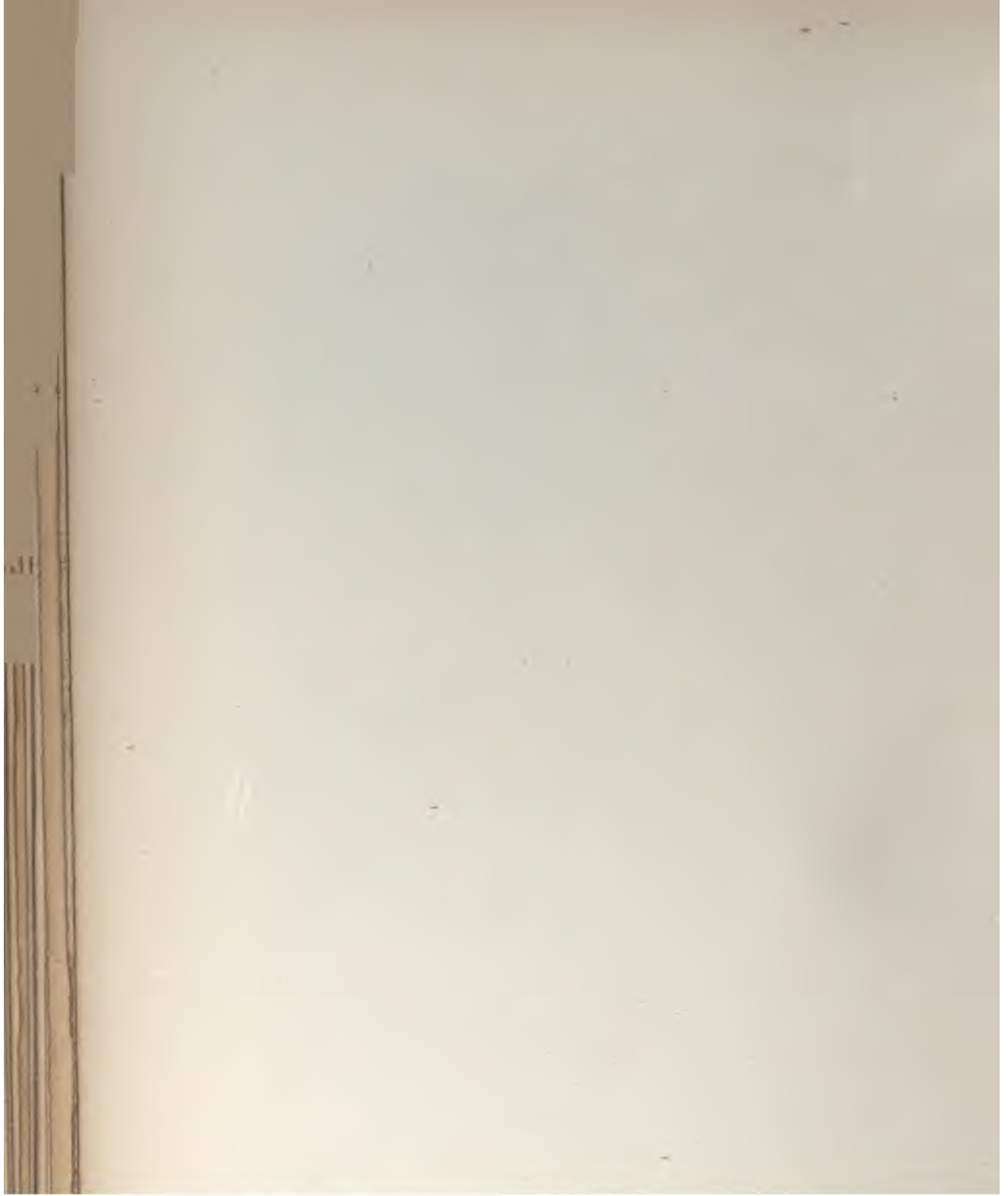
Plate
ACU

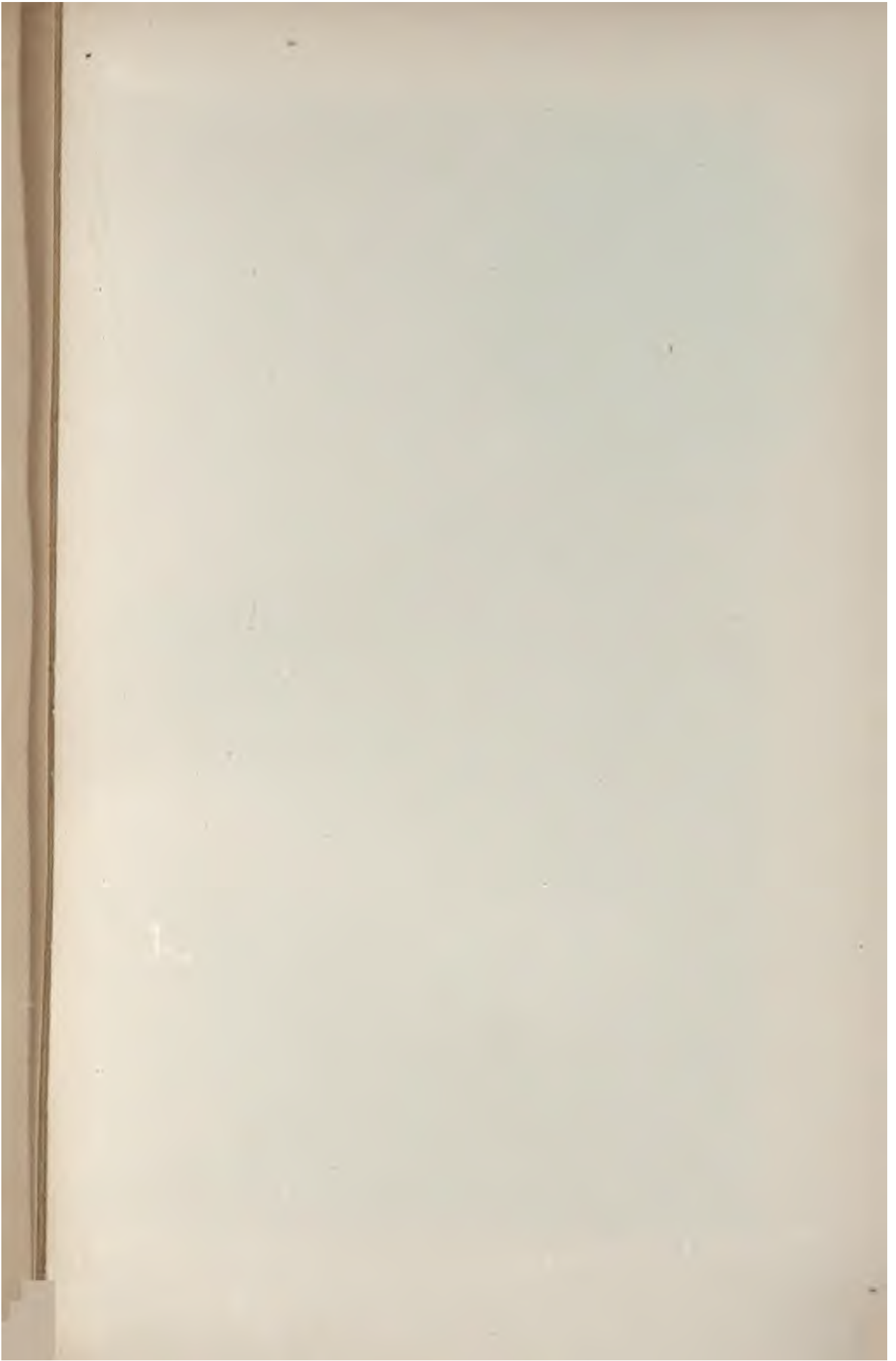


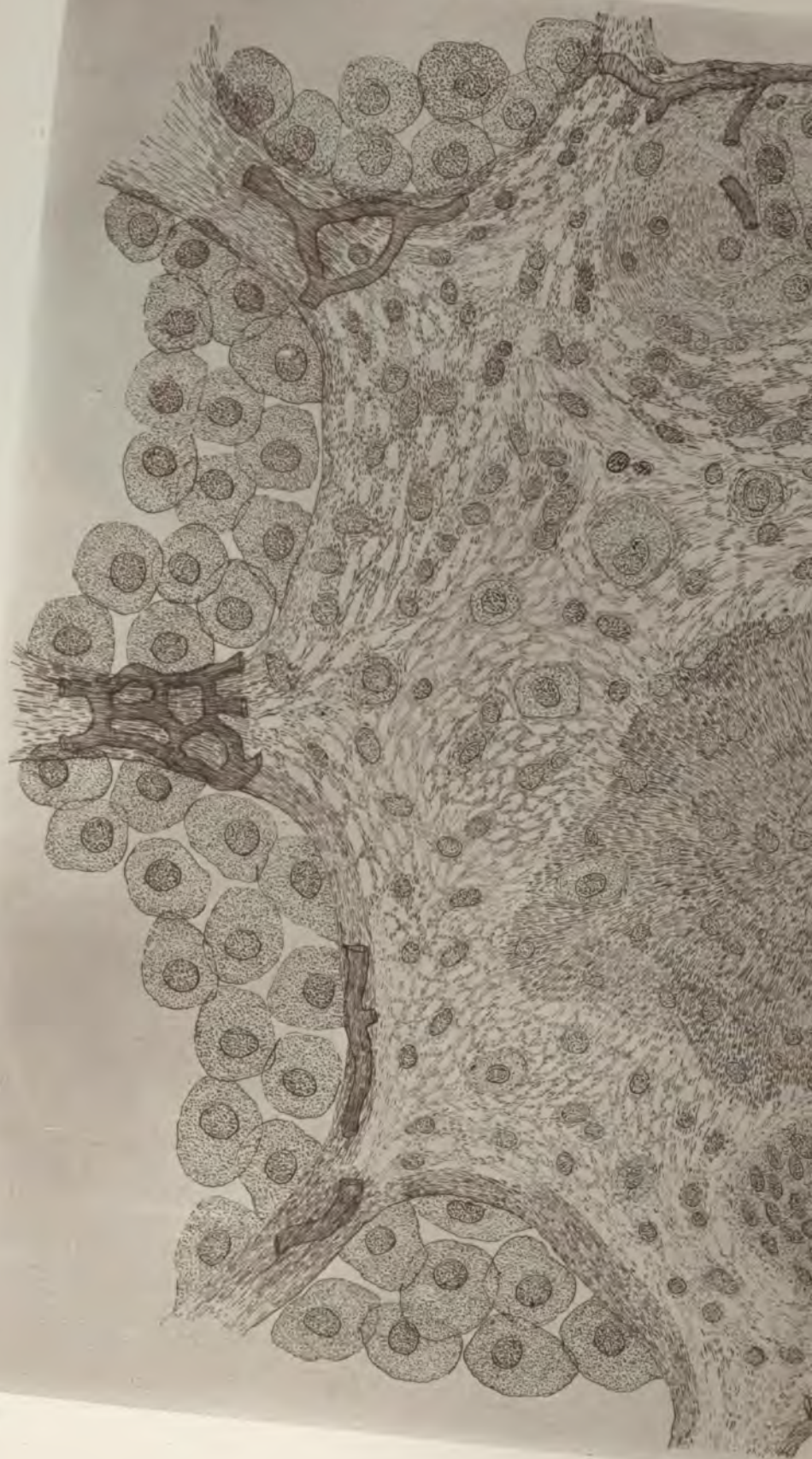
PLATE 100

LXXXVIII. x850

TE PHTHISIS

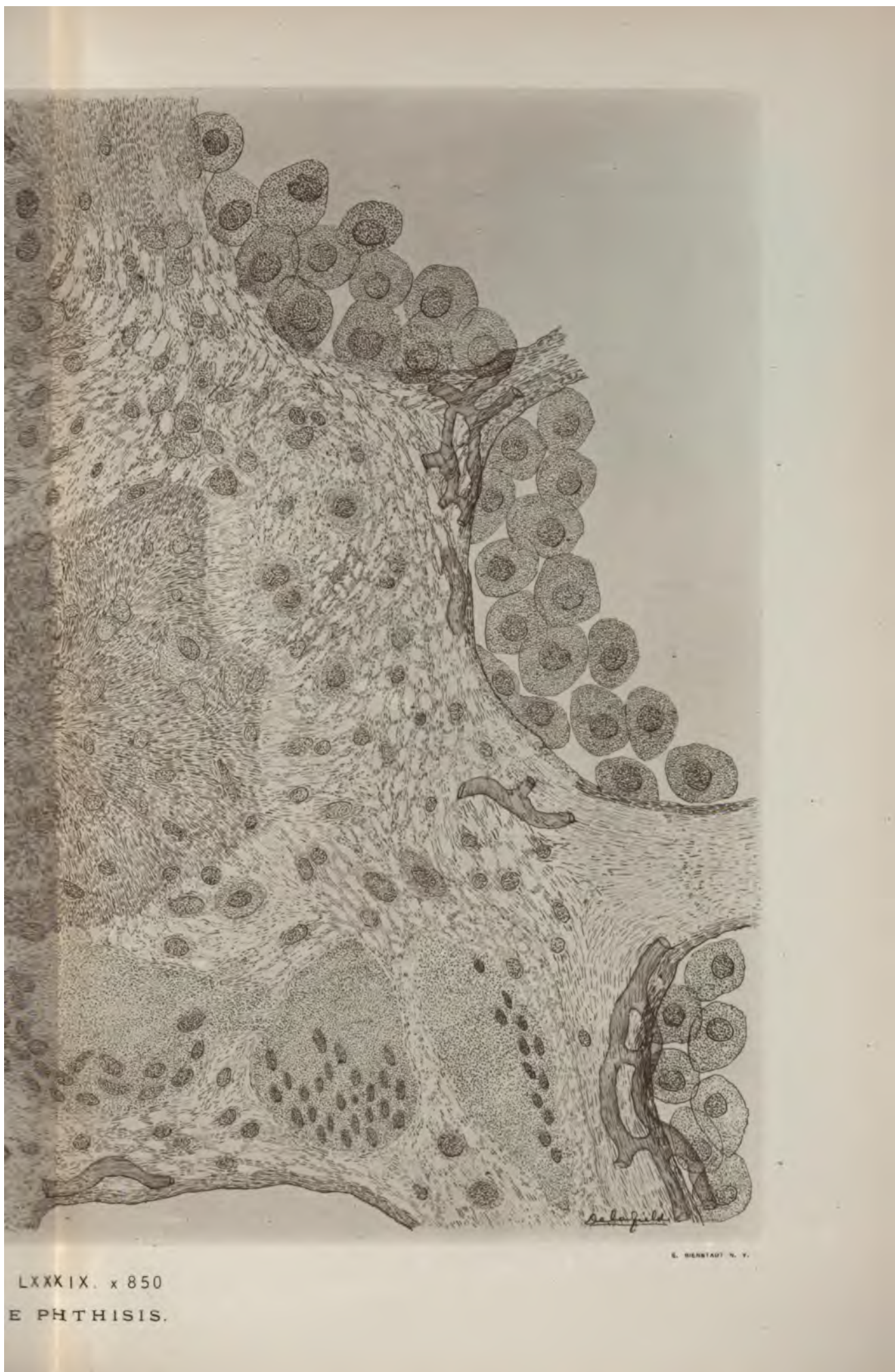






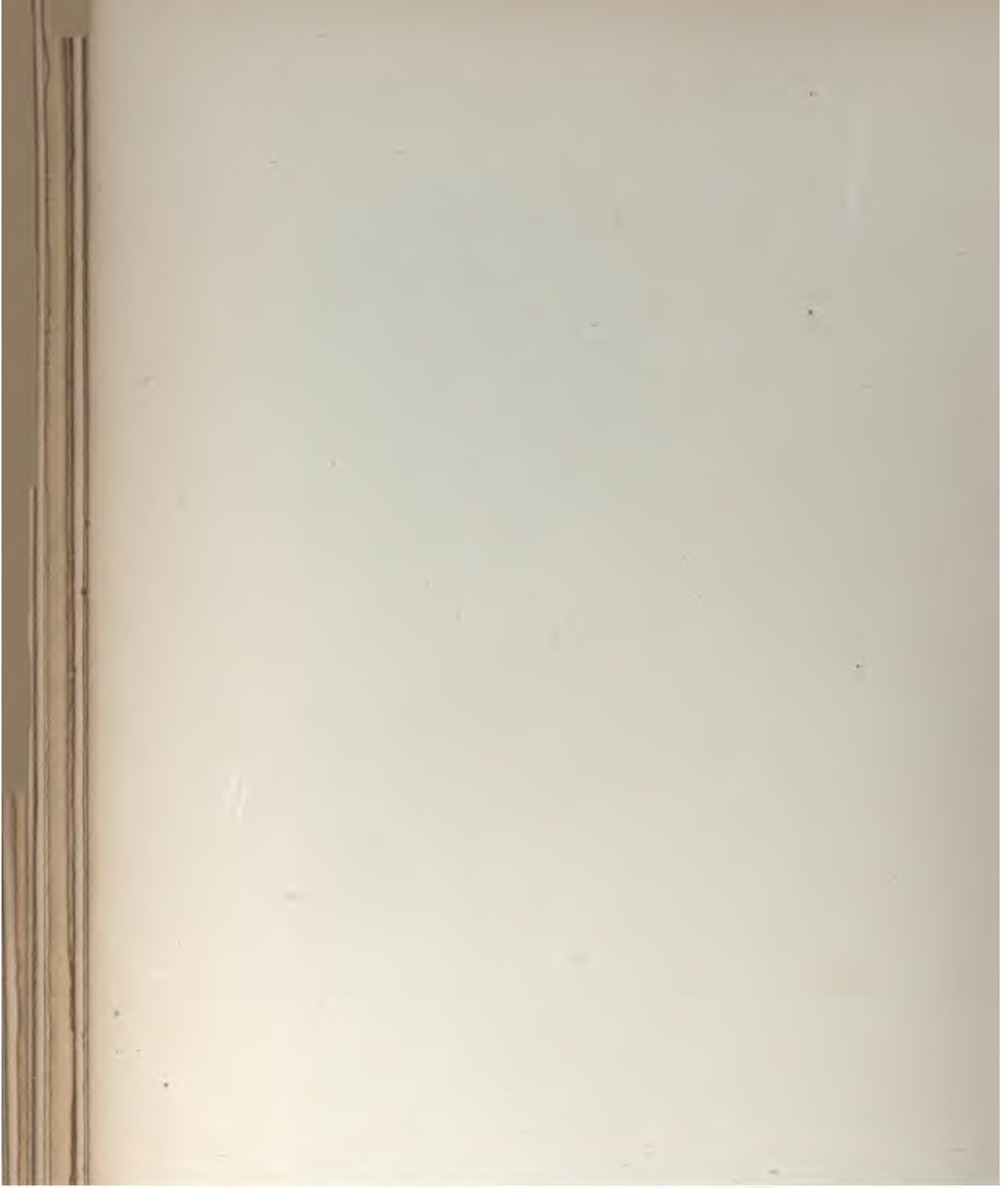
ARTOTYPE,

Plate LXX.
ACUTE PH



LXXXIX. x 850

E PHTHISIS.



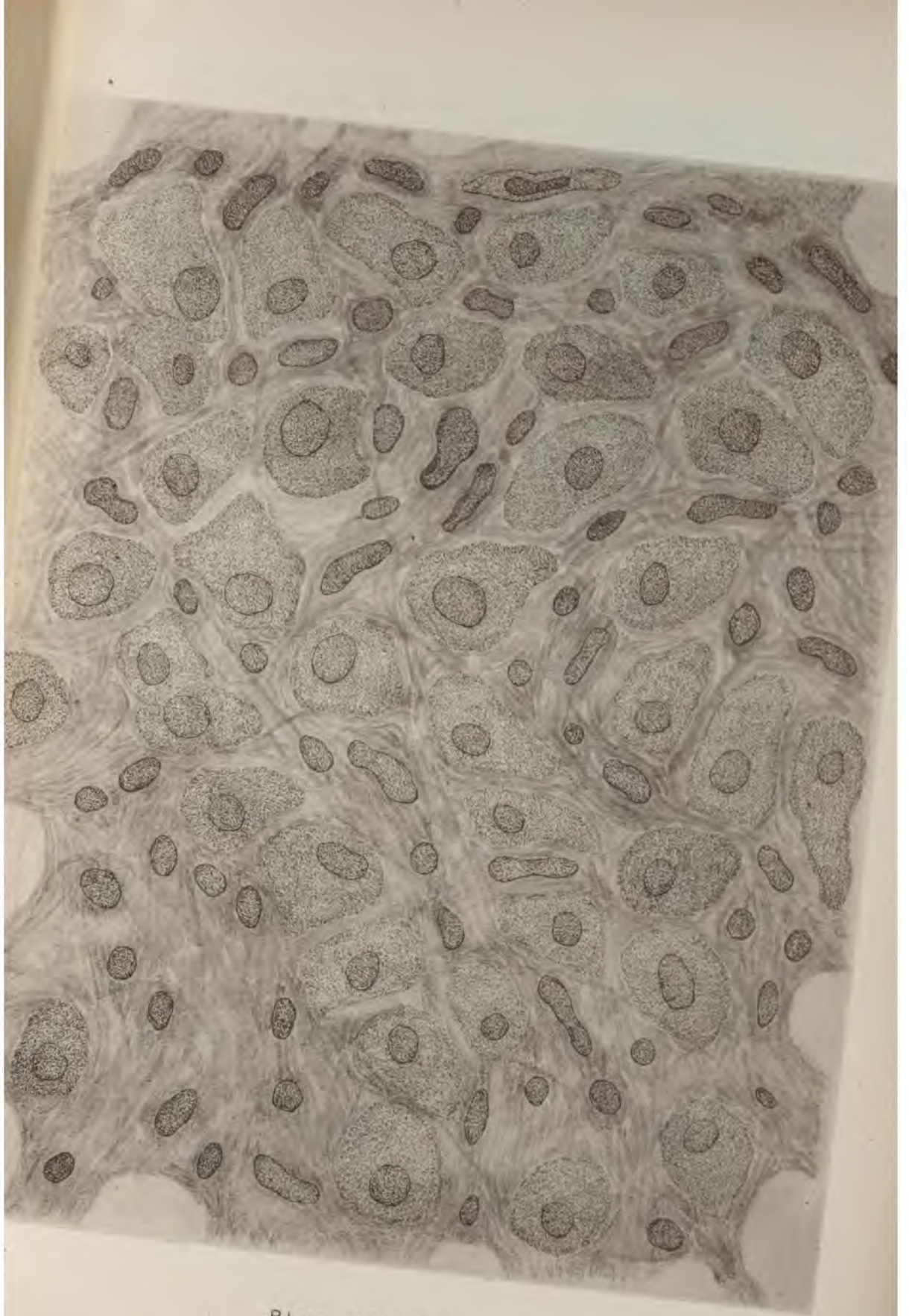


Plate XC. x1500
ACUTE PHTHISIS.

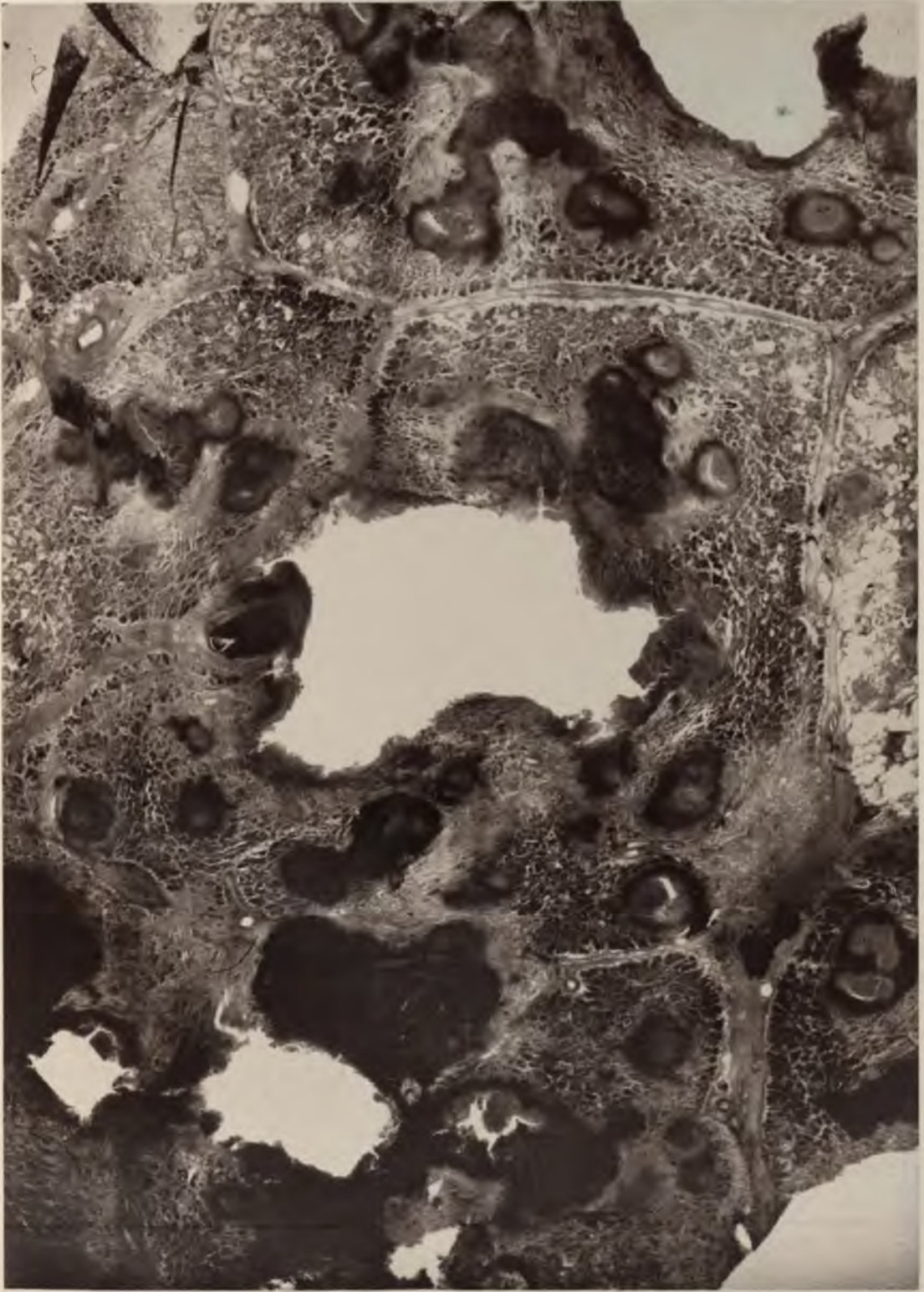
L. DENSTADT M. D.





Plate XCI. x 7

ACUTE PHTHISIS.



ARTOTYPE.

V. BERNHARDT N. Y.

Plate XCII. x 10

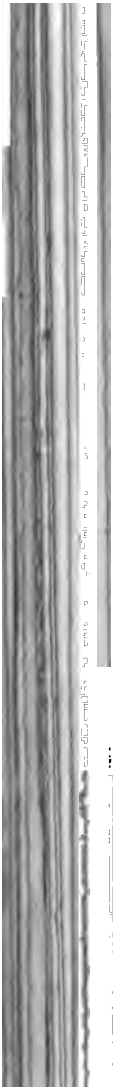
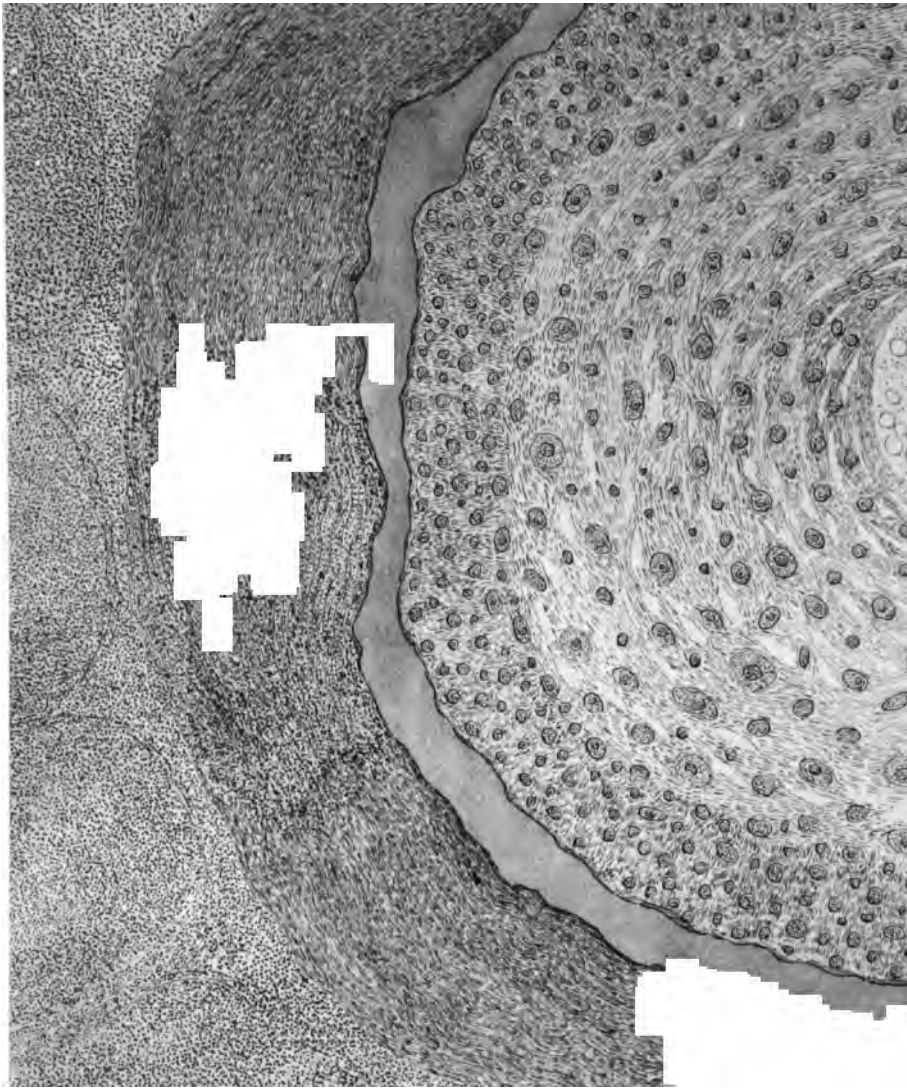
ACUTE PHTHISIS.

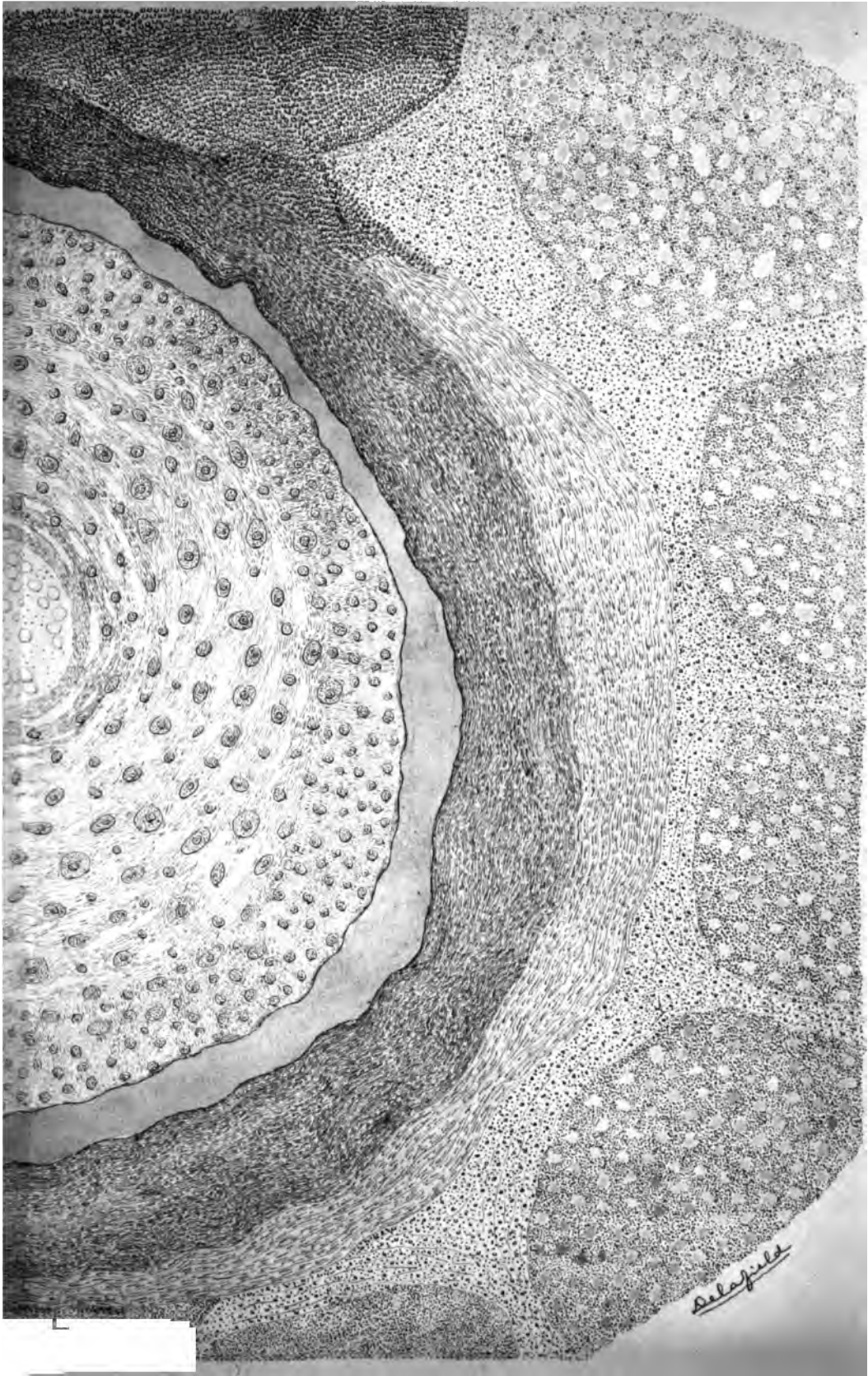


degeneration. It does not involve at once the whole of the centre of the tubercle, but begins first in a few of the elements at the centre and then extends irregularly outward, involving more and more of the nodules.

It seems to me that the only way to account for the formation of these centres of coagulation necrosis is by supposing an occlusion of the branches of the pulmonary artery. The changes are exactly similar to those seen in the white infarctions of the kidneys and spleen; it is generally admitted that the small as well as the large branches of the pulmonary artery are terminal arteries; it has been demonstrated by different writers that the inner coat of the arteries is often changed in phthisis; the different sizes of the areas of coagulation necrosis would correspond with the blocking up of smaller or larger arteries. On the other hand, I have not been able to demonstrate that the arteries are regularly plugged. In some cases indeed I have found a well-marked obliterative arteriitis of the larger arteries such as is seen in Plate XCIII., but that is all.

It seems evident then that in acute phthisis we have a lesion of peculiar characters. It is very far from being a simple broncho-pneumonia with degenerative changes, or an acute miliary tuberculosis combined with pneumonia. The lesions are much more complex. Both the bronchi and the parenchyma of the lung take a share in the morbid process. The lesion seems to be an inflammatory one, the products of inflammation being pus, epithelium, fibrine, and tubercle tissue; but it is complicated by the formation of areas of coagulation necrosis. The tubercle tissue is arranged partly so as to form miliary tubercles and large tubercles, partly as peri-bronchitic nodules, partly as tubercular zones around areas of coagulation necrosis. The prognosis seems to depend mainly upon the number and size of the areas of coagulation necrosis, and it seems probable that most of these are formed at the very outset of the disease. The diffuse pneumonia may undergo resolution, the miliary tubercles and peri-bronchitic nodules do not occupy much of the lung and may be transformed into cheesy material or fibrous tissue; but the areas of coagulation necrosis destroy a considerable part of the parenchyma of the lung and have a natural tendency to soften and form cavities.





CHI 4480

ARTERITIS



EXPLANATION OF THE PLATES.

Many of the plates should be looked at with a magnifying glass.

PLATE I. \times 750.

(Autographic process.)

Flat connective-tissue cells from the surface of the fascia of a dog's leg. In the upper part of the drawing the outlines of the cells are demonstrated with nitrate of silver; in the lower part of the drawing the same cells are stained with hæmatoxylin and eosin, and isolated.

PLATE II. \times 1500.

(Photo-lithograph of a drawing.)

Branching connective-tissue cells from the tendon of a dog's leg. Hæmatoxylin and eosin.

PLATE III. \times 750.

(Photo-lithograph of a drawing.)

Branching connective-tissue cells in the omentum of the rabbit. Nitrate of silver preparation.

PLATE IV. \times 750.

(Heliotype of a drawing.)

The inner surface of the costal pleura of the dog. The endothelial cells have been brushed off from the upper part of the specimen, showing the connective tissue, elastic fibres, and capillary blood-vessels beneath.

PLATE V. \times 1500.

(Photo-lithograph of a drawing.)

In the upper part of the plate, the endothelial cells; in the lower part, the fixed connective-tissue cells of the dog's pleura. Hæmatoxylin and eosin.

PLATE VI. \times 90.

(Photo-lithograph of a drawing.)

Endothelium of the human costal pleura, demonstrated with nitrate of silver.

PLATE VII. \times 90.

(Drawn on wood.)

The lymphatics of the costal pleura of the dog, demonstrated with nitrate of silver.

PLATE VIII. \times 750.

(Heliotype of a drawing.)

The costal pleura of the dog, the lymphatic spaces and fixed connective-tissue cells. Nitrate of silver and hæmatoxylin.

PLATE IX. \times 750.

(Drawn on wood.)

Pleurisy of the dog, produced by chloride of zinc, of twenty-four hours' duration. The cell-growth in the connective tissue just beneath the endothelium; swelling of the old cells and production of new ones. Hæmatoxylin and eosin preparation.

PLATE X. \times 750.

(Heliotype of a drawing.)

Chloride of zinc pleurisy of the dog on the third day. A layer of fibrine, and of new cells stripped off from the surface of the costal pleura. Hæmatoxylin and eosin preparation.

PLATE XI. \times 750.

(Drawn on wood.)

Chloride of zinc pleurisy of the dog on the fifth day; the blood-vessels have been artificially injected from the aorta. A layer of new cells and new blood-vessels stripped off from the surface of the costal pleura. Hæmatoxylin and eosin.

PLATE XII. \times 750.

(Photo-lithograph of a drawing.)

Chloride of zinc pleurisy of the dog, seventh to ninth day. Isolated endothelial and connective-tissue cells from the layer of new tissue on the surface of the costal pleura. Hæmatoxylin and eosin.

PLATE XIII. \times 750.

(Photo-lithograph of a drawing.)

Empyema of the dog, produced by a seton, of twenty-four hours' duration. Changes in the endothelial cells of the costal pleura and production of pus-globules beneath them. Hæmatoxylin and eosin.

PLATE XIV. \times 750.

(Photo-lithograph of a drawing.)

Empyema of the dog, of ten days' duration. Vertical section of the costal pleura; showing new cells, basement-substance and blood-vessels. Hæmatoxylin and eosin.

PLATE XV. \times 750.

(Etched on copper.)

Human empyema. Vertical section of the costal pleura, showing the splitting up of the basement-substance by new cells. Hæmatoxylin and eosin.

PLATE XVI. \times 750.

(Etched on copper.)

Human hydrothorax. Endothelial cells on the surface of the costal pleura.

PLATE XVII. \times 750.

(Photo-lithograph of a drawing.)

Chronic pleurisy with adhesions, human. The surface of the costal pleura, covered with changed endothelium and giant-cells; beneath these the connective-tissue layer of the pleura, with capillary blood-vessels and large connective-tissue cells. Hæmatoxylin and eosin.

PLATE XVIII. \times 90.

(Photo-lithograph of a drawing.)

Tubercular pleurisy. A vertical section of the costal pleura, showing the infiltration of the substance of the pleura with tubercle granula and diffuse tubercle. Hæmatoxylin and eosin.

PLATE XIX. \times 750.

(Photo-lithograph of a drawing.)

The parietal peritoneum of the dog, showing the free surface of the peritoneum, partly covered by endothelium, beneath this the connective tissue with its lymphatic spaces. Nitrate of silver, hæmatoxylin and eosin.

PLATE XX. \times 750.

(Etched on copper.)

The normal human omentum, covered with endothelium, fusiform and branching cells in the connective tissue. Nitrate of silver, hæmatoxylin and eosin.

PLATE XXI. \times 750.

(Photo-lithograph of a drawing.)

Cellular peritonitis, human. Production of new cells on the surface and in the substance of the omentum. Hæmatoxylin and eosin.

PLATE XXII. \times 750.

(Photo-lithograph of a drawing.)

Cellular peritonitis of the dog on the fourth day after an injection of chloride of zinc into the peritoneal cavity. Cell-growth on the surface of the omentum. Hæmatoxylin and eosin.

PLATE XXIX. \times 750.

(Drawn on stone.)

Tubercular peritonitis, human. Section of a tubercle granulum. Hæmatoxylin and eosin.

PLATE XXX. \times 1300.

(Drawn on stone.)

Part of the wall of an air-vesicle of an adult human lung, showing all the cells that could be seen in a hæmatoxylin and eosin preparation.

PLATE XXXI. \times 300.

(Drawn on stone.)

The pneumonia of heart disease (brown, or pigment induration). The walls of the vesicles are thickened, their cavities partly filled with epithelial cells. Hæmatoxylin and eosin.

PLATE XXXII. \times 750.

(Drawn on stone.)

The pneumonia of heart disease. A single air-vesicle. Dilatation of the capillaries, thickening of the walls, production of epithelium.

PLATE XXXIII. \times 750.

(Drawn on stone.)

Acute lobar pneumonia, commencing red hepatization. A single air-vesicle, the vessels artificially injected; the cavity of the vesicle is partly filled with fibrine, pus, and epithelium.

PLATE XXXIV. \times 500.

(Photo-lithograph of a drawing.)

Croupous bronchitis with lobar pneumonia. Vertical section of the wall of a bronchus. A layer of fibrine and pus on the inner surface of the mucous membrane, and another layer of fibrine and pus between the epithelial cells and the basement-membrane.

bronchus. The epithelial cells are swollen and altered; the connective tissue beneath is infiltrated with cells.

PLATE XLII. $\times 22$.

(Drawn on stone.)

Acute miliary tuberculosis. Section of a number of miliary tubercles.

PLATE XLIII. $\times 300$.

(Drawn on stone.)

Section of a single miliary tubercle composed of a group of air-vesicles. The walls of the air-vesicles are infiltrated with tubercle tissue, and their cavities are filled with the same tissue. It is that variety of tubercle tissues in which the cells are large, numerous, and close together in the meshes of the basement-substance. In the surrounding vesicles are an increased number of epithelial cells.

PLATE XLIII. No. 2. $\times 300$.

(Artotype of a drawing.)

Section of a single miliary tubercle composed of a group of air-vesicles. The walls of the vesicles are split up into tubercle tissue, their cavities are filled with the same tissue. It is that variety of tubercle tissue in which the basement-substance is in excess, and the meshes, for the most part, empty of cells. Giant-cells are present. The entire group of vesicles is surrounded by a zone of connective tissue infiltrated with small round cells.

PLATE XLIV. $\times 1500$.

(Drawn on stone.)

Tubercle tissue from a tubercle granulum. The meshes of the basement-substance are filled with polygonal nucleated cells.

PLATE XLV. $\times 300$.

(Drawn on stone.)

Section of a single miliary tubercle, composed of two large tubercle

The blood-vessels have been artificially injected, showing blood-vessels in some of the tubercles.

PLATE LIII. \times 850.

(Drawn on stone.)

Chronic miliary tuberculosis. Part of the wall of an air-vesicle, showing the growth of cells on its surface.

PLATE LIV. \times 90.

(Photo-lithograph of a drawing.)

Chronic miliary tuberculosis. Several miliary tubercles composed of tubercle granula and diffuse tubercle.

PLATE LV. \times 300.

(Drawn on stone.)

Chronic miliary tuberculosis. Diffuse growth of tubercle tissue filling and compressing the air-vesicles.

PLATE LVI. \times 90.

(Photo-lithograph of a drawing.)

Chronic miliary tuberculosis. Old tubercles changed into granular matter and fibrous tissue.

PLATE LVII. \times 850.

(Photo-lithograph of a drawing.)

Chronic miliary tuberculosis. Tubercle tissue between the miliary tubercles.

PLATE LVIII. \times 90.

(Drawn on stone.)

Chronic miliary tuberculosis. A single miliary tubercle, the blood-vessels injected.

PLATE LXVI. \times 850.

(Artotype of a drawing.)

The red hepatization of acute phthisis. A single air-vesicle filled principally with fibrine, the blood-vessels artificially injected.

PLATE LXVII. \times 200.

(Artotype of a drawing.)

The red hepatization of acute phthisis. A group of air-vesicles filled with granules, the blood-vessels artificially injected.

PLATE LXVIII. \times 16.

(Artotype of a drawing.)

Human lung, corrosion preparation. Fragments of several air-passages, and part of a small bronchus.

PLATE LXIX. \times 5.

(Photograph of a specimen.)

Section of a normal human lung, showing the lobules, air-passages, and air-vesicles.

PLATE LXX. \times 300.

(Artotype of a drawing.)

Acute phthisis. A section of an air-passage with air-vesicles. The centre of the air-passage is filled with tubercle tissue, the peripheral portions and the surrounding vesicles with epithelium and fibrine. The blood-vessels are injected.

PLATE LXXI. \times 4.

(Photograph of a specimen.)

Acute phthisis. Diffuse hepatization produced by single air-vesicles and air-passages filled with tubercle tissue, small areas of coagulation necrosis, some of them corresponding to air-passages, and ordinary pneumonia. The plate should be looked at with a magnifying glass.

PLATE LXXII. \times 120.

(Artotype of a drawing.)

Acute phthisis. Section of a miliary tubercle.

PLATE LXXIII. \times (?)

(Photograph of a specimen.)

Acute phthisis. Peri-bronchitic nodules and diffuse pneumonia.

PLATE LXXIV. \times 15.

(Artotype of a drawing.)

Acute phthisis. Section of a single lobule containing isolated peri-bronchitic nodules. The blood-vessels are injected.

PLATE LXXV. \times 4.

(Photograph of a specimen.)

Acute phthisis. Peri-bronchitic nodules, areas of coagulation necrosis, miliary tubercles, and diffuse pneumonia.

PLATE LXXVI. \times 120.

(Artotype of a drawing.)

Acute phthisis. A peri-bronchitic nodule formed principally by a change of the surrounding air-vesicles into tubercle tissue. The walls of the bronchus are infiltrated and its cavity contains inflammatory products. The large blood-vessels are injected.

PLATE LXXVII. \times 120.

(Artotype of a drawing.)

Acute phthisis. A peri-bronchitic nodule formed of tubercle tissue. Cheesy degeneration has commenced around the bronchus.

PLATE LXXVIII. \times 120.

(Artotype of a drawing.)

Acute phthisis. A peri-bronchitic nodule formed of tubercle tissue.

The wall of the bronchus is infiltrated and has undergone cheesy degeneration. The blood-vessels are injected.

PLATE LXXIX. \times 850.

(Artotype of a drawing.)

Acute phthisis. Part of a peri-bronchitic nodule. The wall of the bronchus is unchanged, but it is surrounded by air-vesicles filled with tubercle tissue. The blood-vessels are injected.

PLATE LXXX. \times 5.

(Photograph of a specimen.)

Acute phthisis. Large bronchi with infiltrated and cheesy walls and dilated cavities.

PLATE LXXXI. \times 8.

(Photograph of a specimen.)

Acute phthisis. General dilation of the bronchi with diffuse hepatization.

PLATE LXXXII. \times 40.

(Artotype of a drawing.)

Acute phthisis. A single air-passage in the condition of coagulation necrosis, surrounded by pneumonia.

PLATE LXXXIII. \times 4.

(Photograph of a specimen.)

Acute phthisis. Large areas of coagulation necrosis.

PLATE LXXXIV. \times 8.

(Photograph of a specimen.)

Acute phthisis. Small areas of coagulation necrosis surrounded by tubercle tissue.

PLATE LXXXV. $\times 11$.

(Photograph of a specimen.)

Acute phthisis. Section of a single lobule. Areas of coagulation necrosis surrounded by tubercle tissue.

PLATE LXXXVI. $\times 850$.

(Artotype of a drawing.)

Acute phthisis. A single air-vesicle filled with the products of inflammation in the condition of coagulation necrosis.

PLATE LXXXVII. $\times 300$.

(Artotype of a drawing.)

Acute phthisis. A small area of coagulation necrosis surrounded by tubercle tissue.

PLATE LXXXVIII. $\times 850$.

(Artotype of a drawing.)

Acute phthisis. Air-vesicles from the tubercular zone surrounding an area of coagulation necrosis, showing the manner in which the vesicles are replaced by tubercle tissue.

PLATE LXXXIX. $\times 850$.

(Artotype of a drawing.)

Acute phthisis. An air-vesicle from the tubercular zone surrounding an area of coagulation necrosis, showing the manner in which the air-vesicles are replaced by tubercle tissue.

PLATE XC. $\times 1500$.

(Artotype of a drawing.)

Acute phthisis. Tubercle tissue from the tubercular zone surrounding an area of coagulation necrosis.

PLATE XCI. $\times 7$.

(Photograph of a specimen.)

Acute phthisis. Cheesy degeneration and softening of areas of coagulation necrosis.

PLATE XCII. $\times 10$.

(Photograph of a specimen.)

Acute phthisis. Cheesy degeneration and softening of areas of coagulation necrosis. At the centre of a lobule is a cavity with ragged edges formed by the softening of several coagulation necrosis areas. The surrounding vesicles are filled with inflammatory products.

PLATE XCIII. $\times 480$.

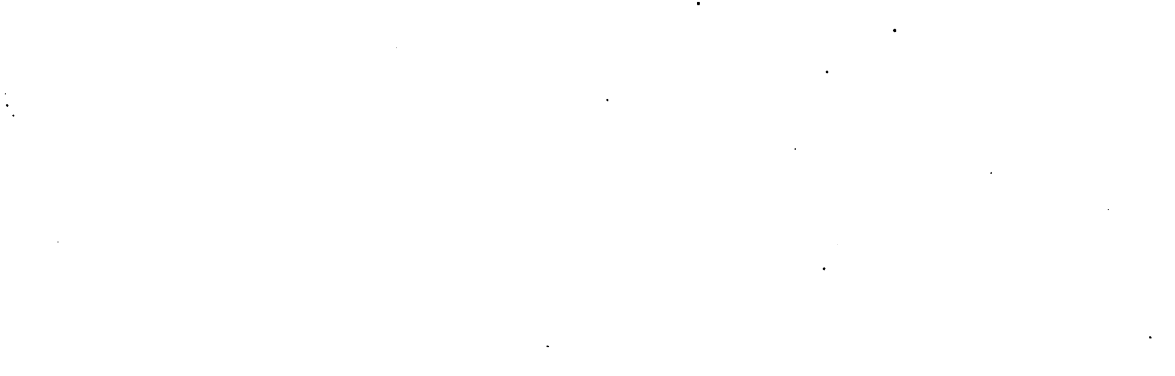
(Artotype of a drawing.)


Acute phthisis. Cross section of an artery situated in the midst of large areas of coagulation necrosis. The lumen of the artery is nearly obliterated by a growth of new tissue.

INDEX.

	PAGE
BRONCHI IN ACUTE PHTHISIS,	105
CHEESY DEGENERATION,	105
COAGULATION NECROSIS,	106
CONNECTIVE TISSUE, NORMAL,	6
CONNECTIVE TISSUE, INFLAMMATION OF,	8
EMPYEMA,	21
HYDROTHORAX,	27
PERITONEUM, NORMAL,	35
PERITONITIS, ACUTE,	37
PERITONITIS, WITH ADHESIONS,	44
PERITONITIS, CHRONIC,	43
PERITONITIS, HÆMORRHAGIC,	46
PERITONITIS, TUBERCULAR,	47
PHTHISIS, ACUTE,	97
PHTHISIS, CHRONIC,	84
PLATES, EXPLANATION OF,	111
PLEURA,	11
PLEURISY, WITH ADHESIONS,	28
PLEURISY, WITH FIBRINE,	15
PLEURISY, WITH FIBRINE AND SERUM,	16
PLEURISY, WITH FIBRINE, SERUM, AND PUS,	21
PLEURISY OF PHTHISIS,	30
PLEURISY, TUBERCULAR,	31
PNEUMONIA,	51
PNEUMONIA OF HEART DISEASE,	53
PNEUMONIA, INTERSTITIAL,	70
PNEUMONIA, LOBAR,	59
PNEUMONIA, LOBULAR,	65
PNEUMONIA FROM PRESSURE,	70
PNEUMONIA, SURGICAL,	65
TUBERCULOSIS, ACUTE MILIARY,	73
TUBERCULOSIS, CHRONIC MILIARY,	87







J25 Delafield, F. 12087
D34 Studies in pathologi-
v.1 cal anatomy
1882

NAME

DATE DUE

